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MEDICINE AND SURGERY AMONG THE AUSTRALIAN ABORIGINES.

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UNTIL the arrival of white men in Australia the natives were decidedly a healthy people, but ever since the first shipload of white settlers landed, in 1788, their health has deteriorated very much and their numbers have rapidly decreased. This deterioration and decrease must be laid at the white man's door, for they are wholly due to his interference with the native tribes. He has introduced those devastating scourges, small-pox, syphilis, and intemperance, with the evils they invariably bring in their train; and probably, also, he has brought with him measles and scarlet fever.

The true nature of a "dispersal" may be gathered from the following. The invading settler locates his stock in some district previously in the sole possession of a tribe. The natives soon see their usual food—roots, seeds, and game—quickly disappear before the squatter and his stock. Impelled both by hunger and revenge, they spear some cattle for food, or, perhaps, while searching for food, some cattle are frightened by them, run away, and are lost. In return for this the settler and his men shoot some natives, who in their turn spear a shepherd or stockrider. The squatter applies to the government for police aid, and half a dozen native police are sent up under a European subinspector.[1]

[1] "So much do the blood-thirsty proclivities of the natives increase by this sort of work, carried on from week to week, that their officers are afraid, as a rule, to ride before their men on the march, lest any whim might lead to their being shot down." —CURR: *The Australian Race*, vol. i. p. 102.

Every native has an intense hatred for every one of his own color who does not belong to his own tribe or the tribes with which he is acquainted, and the police department takes advantage of this, and always sends native police recruited from tribes far from the scene of action. Well mounted, well armed, well supplied with ball-cartridges, the black troopers, hot for blood, are put on the trail. Then follow tracking, surprise, and shooting from beyond spear range, which result in the death of many men, the capture of the women, the gratification of savage lust, and a report from the subinspector to head-quarters that the offending tribe has been "dispersed." After several such experiences, and most of the young men have been butchered, the demoralized natives realize the impossibility of winning in the unequal fray, and the tribe—or rather its remnants—are allowed to "come in" to the settlers' homestead, and the struggle is over. In the end syphilis finishes what the rifle began. To what an extent this has happened may perhaps be realized when it is stated that the last Tasmanian died in 1876, and that in Victoria the few natives still remaining are at the government aboriginal stations, while the "dispersal" process is still going on in other Australian colonies.

DISEASES OF THE ABORIGINES.

The ailments which have been observed among the aborigines are, in alphabetical order, ague, apoplexy, asthma, boils, bronchitis, cancer, cataract, cholic, colds, diarrhœa, diseases of the stomach and intestines, dropsy, dysentary, dyspepsia, ear-diseases, epilepsy, fever, fistula in ano, heart-disease, hydatids, hydrocephalus, hydrothorax, influenza, insanity, intemperance, kidney-disease, leprosy, liver complaint, malaria, measles, neuralgia, nostalgia, ophthalmia, paralysis, phthisis, pleurisy, pneumonia, rheumatism, scarlatina, scrofula, scurvy, skin-diseases (such as favus, impetigo, itch, mange, porrigo, ringworm), small-pox, sore throat, syphilis, tabes mesenterica, tuberculosis, and toothache.

The most fatal of all these to the blacks are those introduced by the white man,—viz., epidemic small-pox, measles, and scarlet fever, also the non-epidemic, but not less deadly, syphilis and intemperance. The wearing of clothes, too, during the day (they always take them off at night) has caused a great extension of chest-diseases and of tuberculosis. "In infancy," says the Rev. George Taplin, "the tuberculous diathesis shows itself frequently in the form of hydrocephalus; in childhood it very often manifests itself in tabes mesenterica; later it often appears in the form of induration and ulceration of the glands of the neck, and in this form is generally cured, the person becoming healthy afterwards; but its most frequent and most fatal form is that of tubercular consumption. Any accident to the chest seems to lead to the deposition of tubercle."

Proximity to the towns leads to intemperance, and intercourse with the whites to venereal diseases. Farm- or town-life often brings on nostalgia or home-sickness, which causes the patient to mope, and if, while in this

state, he catches even a slight cold, lung-disease develops and death follows. In nostalgia, cod-liver oil and cantharides have been found useful. Leprosy, again, has been introduced by the Chinese, Japanese, Malays, Afghans, and other Asiatic races, and also by the South Sea Islanders on the sugar plantations.

INTRODUCTION OF SMALL-POX.

The history of small-pox in Australia is not without interest. Dampier, keen observer though he was, saw no pock-marked natives on his visit in 1698. Neither did Captain Cook during his stay a Botany Bay in 1770. Yet in the parts they visited pock-marked old men have been common for decades. The next arrivals in Australia were the first convicts, who reached Botany Bay in January, 1788, and left for Port Jackson, now Sydney Harbor, a few days later. As the English left the bay, two French ships under de la Perouse sailed in and remained in the bay till March 10.

In April, 1789, fifteen months after the arrival of the English ships, and thirteen months after the departure of the French vessels, a violent epidemic resembling small-pox mowed down the natives as corn is mowed by the scythe. Many denied that it was small-pox, on the grounds that had it been such it would have become epidemic among the whites, who tended and fed the sick; that when whites were attacked the contagion did not spread among the settlers, and that the wild dogs who devoured the abandoned corpses of the natives came to no harm. Those, on the other hand, who considered it small-pox pointed out that the whites were mostly inoculated; that in two cases the disease had attacked whole families of whites; that the symptoms and after pitting were the same as those of small-pox; that those who were already inoculated were not attacked, and that immediate inoculation modified very much the severity of the attack.

The small-pox party having gained the day, the English settlers patriotically laid the blame of its introduction on the ships of de la Perouse. But E. M. Curr, in his work, "The Australian Race" (1886), proved beyond a doubt that there was small-pox on board of the transport "Alexander," one of the ships of the first English fleet, and that, on landing, clothes from the men on the ships had been given to the natives; so the blame for its introduction on the east coast lies with the English. On the north and northwest coasts, however, it was undoubtedly introduced by Malay pearl and trepang fishers. Confirmation of these facts is found in the traditions of the aborigines, those of Central Australia averring that the disease came from the eastward, those near Adelaide declaring that it came to them down the Murray River, and those of West Australia affirming that it came from the northward.[1]

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[1] Further confirmation is found in the fact that in the subdialects of a group of related tribes the words denoting small-pox are generally quite different, or else there is no special word at all, that for "nasty" or "poisonous" being pressed into service. Compare newspaperese "loathsome disease" = syphilis.—J. S. R.

SPREAD OF SYPHILIS.

The rapid spread of syphilis may be attributed to many causes, such as the white convicts' lust, the absence of white women in the bush, the native superstition that a young woman whose first intercourse was with a white man would bear afterwards exceptionally strong and healthy black sons, and the customs of exchanging wives, of handing over a wife, when going on a journey, to an unmarried male friend, and of providing visitors, both black and white, with female bedfellows.

NATIVE "DOCTORS."

Like other savages, the Australian aborigines in their natural state are firm believers in their native "doctors," who, as elsewhere among uncivilized people, are usually arrant frauds. Dances, incantations, and legerdemain are their chief stock in trade. A "doctor" will examine a patient, feel the parts affected, rub them, suck them, and then pretend to have discovered the cause of the sickness. Thereupon he goes away for a few minutes, returns, resumes his rubbing and sucking, and presently, after a little manœuvring, seems to produce from the affected part pieces of stone, bone, stick, charcoal, or even string, and triumphantly announces that these have been the cause of the trouble. This reminds one of the Chinese tooth-ache-curers, who produce little bunches of worms(?) from decayed teeth. But the strangest thing about the business is that a "doctor," though knowing full well the fraudulent nature of the whole procedure, will, when ill, call in another "doctor" to practise upon him! Fancy one "medical clairvoyant" calling in another to diagnose his sickness! Yet it is certain that cures often follow all this nonsense. Probably "faith" and nature are the true healers in such cases.

ABORIGINAL THERAPEUTICS.

Some tribes, however, proceed in a much more rational manner, employing the lancet, bandages, splints, poultices, plasters, water-compresses, lavage, massage, bleeding, vapor baths, emetics, astringents, etc. Here are a few examples of their methods of treatment:

Boils.—When hard, boils are treated with a decoction of wattle-bark (*Acacia pycnantha*); when, after this treatment, they are obstinate, a poultice of boiled marshmallow (*Althæa officinalis*) is used; if they soften without breaking, they are pricked with a bone lancet. When the boil comes on a limb, a ligature is tied above to check circulation.

Colds.—Sometimes the treatment for these is cold water within and warmth without; sometimes an infusion of wild mint (*Mentha laniflora*) is given internally, and, as an aperient, an infusion of wild lavender-tree leaves.

Colic.—Sometimes friends press and knead the stomach with their feet; sometimes hot eucalyptus leaves or hot stones are applied.

Dysentery.—A decoction of wattle-bark is sometimes taken inwardly for dysentery, while wattle-gum is chewed all day, and pills of wattle-bark and gum are taken night and morning. Sometimes the patient lies on his back with his head turned sideways, and a native doctor presses the upturned ear with his foot till the pain causes water to gush out at the eyes, and occasionally this effects a cure.

Dyspepsia.—Cold water is given inwardly and outwardly.

Ear-Diseases.—These are treated with juice from the roasted fruit of the red eugenia (*E. australis*).

Fever.—Sometimes fever is treated with an infusion of the leaves of *Eucalyptus tetrodonta;* at other times by means of continual bathing. The latter treatment is generally fatal.

Headache.—A tight bandage, often of green hide, is bound round the head; or the head is held over a fire of green twigs; or smoked green leaves are applied hot; or blood is let from the temples, scalp, or forearm.

Lung-Disease.—If the blood-spitting becomes serious, a native "doctor" presses with his feet or even jumps on the patient's body; the natural result is hemorrhage and death.

Neuralgia.—A poultice of fruit of the red eugenia (*E. australis*) is applied to the affected part.

Ophthalmia.—Eye-troubles are treated with the juice of the pig-face (*Mesembrianthemum æquilaterale*), or by means of repeatedly washing with water.

Rheumatism.—The skin is scarified; or hot stones are applied to the part; or it is poulticed with boiled marshmallow (*Althæa officinalis*); or a tight bandage is bound over the affected part; or hot ashes are rubbed in; or the patient is buried neck-deep in the sand of the sea-shore; or the patient is laid on a wicker stage, under which heated stones covered with wet water-weed ("pinngi") are placed, and the whole is covered in with rugs; in fact, an improvised vapor-bath is given.

Skin-Diseases.—Affections of the skin are treated by bathing and attention to cleanliness; or by an ointment of grease, red ochre, and a decoction of wattle-bark; or by scratching the parts with shells; or by bruising the pustules; or by opening the pustules with a pointed stick or piece of bone.

Small-pox.—The juice of a leafless creeper, name unknown, is used as a therapeutic agent. Whether it is really efficacious is not known.

Sores.—Sores are sucked by a native "doctor"; or regularly washed with water; or a ligature is tied above; or hot earth or ashes are bound on.

Venereal Diseases.—A very strong decoction of wattle-bark is used in all venereal diseases, and has been found very efficacious even in severe attacks of syphilis.

Toothache.—The gums are bled.

Snake-bite.—The wound is sucked; or a ligature is applied above and below, and an artery is opened, or else numerous incisions are made between ligatures, and the sufferer freely bled.

Fracture.—Fractures are bound up in bark splints, and recovery takes place in a short time; if it is a comminuted fracture of limb-bones, and it is impossible to save the damaged part, it is inserted in a hole in the ground, which is filled with earth to a point a little above the injury; live embers are then placed round, and the injured part thus burned off.

Burns.—Burns are first greased with melted fat and then dusted with red ochre.

Wounds.—Eucalyptus gum or bark steeped in urine is employed as a dressing, or the wound is cauterized; or it is sucked and then either poulticed with chewed or steeped eucalyptus leaves, or plastered with clay, earth, mud, or hot ashes.

Spear-wound.—When the spear remains in, the shaft is broken off short and the barbed part is pulled through; or the spear is twirled round till the wound is large enough for it to be drawn out easily.

Sting.—Hot leaves are applied.

Bleeding.—Hemorrhage is stopped by binding with spongy bark; if the bleeding is from a limb, a ligature is applied above.

The aborigines in their natural state recover very rapidly from wounds and fractures, even when they are of the most serious kind. A fractured collar-bone has been restored to working order in three days, and no inconvenience suffered then or afterwards. Their power to endure pain, labor, and hunger is astounding.

OPERATIVE SURGERY.

Beyond the use of ligatures, bandages, splints, the lancet and the cautery, the blacks know practically nothing of surgery. Their nearest approaches to operative surgery proper are connected with ornamentation of the body, coming-of-age rituals, Malthusian purposes, and incantation ceremonies.

For ornamental purposes, they make a series of parallel scars on the chest, back, upper arm, and the thighs in the case of both sexes, and on the abdomen and round the breasts of females. In most tribes, also, the septum of the nose is pierced with a pointed bone or stick, and the hole is kept open with a quill, to allow of the later insertion of some foreign body, such as a feather, bone, or twig. The parallel wounds are cut with sharp flints, quartz slivers, or shells, or are burned in with sharp hot stones. They are then filled in with earth, charcoal or ashes, and the edges are kept from uniting for some weeks. When healed, the scars are about the length of the little finger, and nearly half an inch high. They resemble nothing so much as rows of large, well-gorged leeches.

Coming of age is marked by knocking out, in various ways, either one or two incisor teeth. The gum round the tooth is first loosened, and then the tooth is made free by placing a stick against it, chisel-fashion, and using a light club as a mallet. Sometimes wedges are fitted in on each side, and then the tooth is loosened as before or the wedges themselves are alternately hammered till the desired end is accomplished. Sometimes the tooth is

made loose with a forked stick used as a lever. The tooth, when loose, is removed by the operator's fingers, and the bleeding is stopped with damp clay or a fire-stick cautery, or else it is allowed to continue.

Circumcision has two ends in view. It is done with a jagged flint at coming of age to test the lad's power of enduring pain, but it also serves another purpose, in that for some time it prevents the young men from intriguing with the young wives of the old men.

Malthusian operations are two. In some few tribes the nipples are cut off the breasts of the girls to prevent them suckling children. In very many tribes what is known as "the terrible rite" is practised on most of the young men, being omitted only at times when the tribe, from sickness or warfare, has been much reduced in numbers. The patient is taken into the bush and spread-eagled on his back; one man holds each limb down firmly, and a fifth sits on the patient's chest. The operator then lays the penis flat on a piece of bark, slits up the urethra from meatus to base, and then binds the organ up with bark in such a way as to effectualy prevent closing. The patient is thus practically, if painfully, rendered sterile.

Incantations are often accomplished with woundings. For example, in the spell to make wild fowl lay plenty of eggs, the men of the Dieri tribe sit in a ring singing an obscene song, and at the same time piercing the scrotum over and over again with the sharpened fibula of a kangaroo. Though this often lays them up for a fortnight or so, they exhibit no signs of pain while the ceremony is going on. During a similar spell to make iguanas plentiful, they pierce their ears several times with leg bones of the emu.

The *amputation* of one or more of the joints of a girl's little finger is done in early childhood by the mother tying a ligature of twine or strong spider's web round the finger, and leaving it there till the joint or joints drop off. The reason for doing this is that the blacks have an idea that the normal little finger is in the way when a woman is rolling up fishing-lines.

In cases where surgical operations involve great pain, it is sometimes relieved by iguana fat.

OBSTETRICS.

As a rule, the aboriginal woman suffers little pain or inconvenience from parturition. During delivery the woman kneels with thighs wide apart, and sitting on her heels, her feet being laid flat on the ground, soles uppermost. One woman squats behind the parturient, and with arms round her waist forms a support for her back. A second midwife does what necessity requires. I have read of women being delivered while standing against a tree. This, however, must refer to cases of sudden labor in which a woman, being alone, squatted (they never *stand*) with her back against a tree, which replaces the support usually given by the midwife.

Though occasionally denied, it is well known to bushmen that twins and triplets are as common among Australian aborigines as among Europeans. It is remarkable that the children are at birth nearly as white as Europeans,

so that the natives sometimes doubt whether they are of pure blood or not. In such cases the form of the nose decides. Half-caste children have occasionally light-colored hair and European features; in fact, it is commonly noticed that the half-caste boy favors the father much more than the mother.

Abortion is occasionally practised, being brought about by pressing or thumping the abdomen. Also, the excessive use of alcoholic drinks lowers fecundity and tends to miscarriages, and prostitution causes sterility, while an excessive use of tobacco causes the infants to be abnormally fat and bloated at birth; such children invariably die.

Count Strzelecki and others have stated that an Australian woman, after having a child to a white man, is ever after sterile to a man of her own race. Not only is such a theory absurd on the face of it, but it is contrary to the experience of bushmen throughout the continent. Brough, Smyth, and Curr have set the question at rest by proving numerous specific cases, not only of black children following a half-caste, but of black, followed by half-caste, followed by black; also at least one case is known of black, half-caste, black, half-caste, black. That case alone shatters Strzelecki's theory at a blow.

A SPECIAL DISEASE.

One disease seems peculiar to the Australian blacks. The stomach becomes perfectly hard, and gradually enlarges enormously. The body wastes to a skeleton, while the appetite is voracious, with an especial craving for meat, though the patient can eat but little at a time, and the food does not seem to nourish. Great debility ensues, and, after many months of misery, the relief of death. Many medicaments have been tried but without success. As no *post-mortem* has ever been held on such a case, even the knowledge derived from anatomizing a case is wanting.

SYPHILITIC SPINAL PARALYSIS.[1]

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THE chief interest in regard to syphilis in the affections of the nervous system lies in the forms it can assume,—in the present case in the cord,—

[1] Dr. Sottas has presented to the public an exhaustive work on syphilis of the spinal cord in the light of knowledge of the present day. There has been no attempt made to do full justice to a book of four hundred and fifty pages in a translation so abridged, but the endeavor has been to offer the chief ideas of the author. The entire second part, devoted to the histological and clinical examination of personal cases, and to the study of those borrowed from other writers, has been left untouched. The three cuts were kindly loaned by the *Revue Neurologique*. W. G. SPILLER.

and in the influence it is capable of exerting in the development of spinal diseases. The first problem only is herein treated, and such systemic troubles as tabes are left untouched.

If the reality of syphilitic myelopathies is acknowledged, it is from a conviction largely due to clinical observation, and on a clinical foundation the syphilitic nature of an affection of the nervous system can be affirmed only by the reasoning *post hoc, ergo propter hoc.* It is true that in certain circumstances this induction has a peculiar value. If, for example, lumbar pains, trouble in micturition, later a more or less rapid weakness of the legs, yielding soon to symptoms of spastic paraplegia, appear in a young person, syphilitic for some months or a few years, it is natural to think of the influence of syphilis, and if no other cause of lesion of the cord, as Pott's disease or traumatism, is evident, this supposition is almost a certainty. But there are other cases where the diagnosis rests on far less precise facts. If the paraplegia occurs in an individual advanced in life, syphilitic for a long time, can one affirm the syphilitic nature of the affection, or is it a paraplegia of unknown origin which has occurred in a syphilitic person? The difficulty very often cannot be settled during the life of the patient. Even at the autopsy it is not always easy to establish precisely the specific nature from the anatomical condition.

The uncertainty increases in regard to certain systematized forms of myelitis, as tabes, which many writers consider to be due either solely or frequently to syphilis.

Are there, in correspondence with the different clinical manifestations of syphilis, special anatomical alterations which permit a classification of these forms? We will first endeavor to ascertain if there are specific lesions of syphilis, and what tissues in the cord can be involved.

As we are ignorant of the nature of the syphilitic infectious agent, we have as a guide only the study of the histological reaction of the tissues involved. In reasoning by analogy we compare the action of the syphilitic virus with that of other infectious agents which produce lesions with a tendency to the formation of nodules, as in glanders, tuberculosis, etc. The specificity of the tubercle has been established only by the isolation, the culture, and the inoculation of the bacillus of Koch. Is the tubercle the sole mode of reaction of the tissues to this bacillus? Cannot this agent produce modifications of the connective tissues other than the tubercle? can it not affect the more essential elements of the different parenchymata? The same questions arise in regard to syphilis, when they are more obscure still, for the reason given.

According to the general opinion the syphilitic virus exerts its influence chiefly on the tissues derived from the mesoderm, especially on the bony and connective tissues and the lymphatic and vascular systems. It is possible that the differentiated elements which constitute the noble tissue of the different organs may be equally directly affected, but this action has

never been actually demonstrated; and until proof to the contrary, the parenchymatous lesion may be considered secondary.

The mode of reaction of the affected tissue has a close affinity to ordinary inflammation, whatever may have been the date of the infection, and here the distinction of secondary and tertiary periods has no foundation. The process of irritation is always the same: it consists in a hyperplasia of connective-tissue cells, of the presence of lymphoid cells, and of a multiplication of those elements which take part in every inflammation.

This inflammatory hyperplasia is often presented in a special form. It can give rise to the formation of specific granulomata, of which the structure has permitted the establishing of the relation between syphilis and the other infectious maladies with nodulary formation.

The importance of this neoplasia is variable, and there are all the intermediate stages between the infiltration more or less conglomerate, which may be regarded as the microscopic (miliary gumma) and the macroscopic gumma. But this cellular hyperplasia is not necessarily circumscribed, this specific character can fail, and there is then a diffuse infiltration.

The evolution of the gumma may be divided into two periods: the first is characterized by the hyperplasia of the cells of the connective tissue, or of similar tissue; in the second, the elements multiply, crowd upon one another, and diminish in volume, while the cells of the central part become granular, atrophy, and the centre of the tumor undergoes caseous change. This tendency to retrograde transformations (fatty and cheesy degeneration) is perhaps peculiar only to large gummata. It is not illogical to consider it as resulting only from the condition of the tumor itself, of which the elements produce by their crowding together an obstacle to the nutrition of the central part.

Have the diffuse gummatous infiltrations the same tendency to this necrobiotic change, or does this tissue, well nourished, remain always vigorous, and tend to become sclerotic? We will find examples of these two forms of evolution outside of the gumma properly so called.

Can syphilis provoke the sclerosis from the beginning? In other words, is it possible to recognize a syphilitic origin for sclerotic lesions not accompanied by diffuse or conglomerate gummatous infiltrations? This is a question still disputed, and especially by the partisans and adversaries of the syphilitic nature of certain systematic affections of the cord.

We have indicated rapidly the modes of reaction of the connective tissue in the presence of the syphilitic infectious agent, but this element is not the only one affected: the predilection of the virus for the vascular tissues is well known, and the vascular alterations are now counted among the manifestations, so to speak, constant in the anatomical modifications produced by syphilis.

Here, as elsewhere, the process lacks anatomical characteristics; it is an infiltration of ordinary aspect, of which the point of departure is still disputed. Certain authors (Heubner, Gerhardt, Litten) regard the internal

tunic as the point of departure of the inflammation. Others (Köster, Lancereaux, Friedländer, Baumgarten) think that the process begins in the adventitia, the other coats being only secondarily affected. Rumpf believes that the thickening of the arterial walls is due to the inflammation of the vasa vasorum, and that the infiltration arises in the media, where the capillaries are the most numerous.

In the nervous centres, and especially in the cord, the syphilis affects primarily only the connective, lymphatic, and vascular tissues.

Whatever may be the tissue involved the inflammation is of the same nature, and there is no real difference between the gumma and the infiltration of the meninges or vascular walls.

It is important to determine the frequency of these localizations, and their relative importance in the determination of the lesions of the nervous parenchyma. We believe that the vascular lesions have a preponderance in importance in the cord, and it is this we attempt to prove in the present work.

Most writers describe two forms of the diffuse syphilitic affection of the cord in relation to the intensity of the symptoms and the rapidity of their evolution: the acute and chronic syphilitic myelitis. The chronic form is the better known, and is often called syphilitic spastic paraplegia, because of the preponderance of spastic symptoms in the inferior members. The acute form, much less known, has been described as assuming the form of an acute diffuse central myelitis. It has always been completely separated from the chronic form, clinically and anatomically, but it is doubtful if there is such a difference between the two, and it is more probable that they arise from an identical process, and that the different clinical appearances are due to variation in extent and intensity of the same lesion. The probability of this is increased by numerous intermediate forms. It is not uncommon to observe the syphilitic paraplegia begin abruptly, and afterwards yield to a persistent spastic paraplegia of a chronic nature. Or in the course of slow evolution, acute attacks occur which aggravate the situation, and rapid death may be caused by such an attack. In still other cases after a period of fluctuation the symptoms increase progressively to a fatal termination. Although these forms may indicate a purely spinal location, the symptoms of cerebral syphilis are often added.

The authors have divided the syphilis of the cord in three groups:

The first comprises the osseous affections, which have their point of origin in the vertebræ and involve the cord secondarily.

In the second are placed the lesions of the meninges, represented by the gumma, the diffuse infiltration, or the fibrous thickening of these envelopes, especially of the pia mater.

In the third category are found the lesions of the cord proper,—myelitis,—but here the confusion is great, and one finds sometimes softening of the cord, sometimes sclerotic induration or embryonic infiltration, and this confusion is not lessened by the employment of so vague a term as mye-

litis. It would be possible to place in a fourth class the cases where the vascular lesions preponderate. These lesions were formerly overlooked, but the analogy with similar lesions in cerebral syphilis has been recently more emphasized. In all the types, gumma, meningitis, myelitis, the vascular alterations are considerable.

The division between the lesions of the pia and those of the cord itself, made for convenience, has no real foundation, for alterations of the nourishing membrane of the cord, however little pronounced, cannot exist without provoking trouble of nutrition and alteration in the cord itself. The lesions of the nervous parenchyma are always subordinate to the primary modifications of the nourishing system of the cord, meninges and vessels.

SYPHILITIC LESIONS OF THE VERTEBRÆ.

Formerly the majority of cases of syphilitic paraplegia observed in syphilitic persons were regarded as due to lesions in the vertebral column, and following the description of the authors two forms were presented: an osseous caries developed under the influence of the syphilis, which by the intermediation of the pachymeningitis produced the paraplegia; and an osteo-periostitis, followed by the development of an exostosis, which compressed the cord.

The syphilitic form of Pott's disease was first described by Portal in 1797. The important *rôle* ascribed to the exostosis is evident in reading the accounts of the paraplegia ascribed to syphilis, and about the middle of the present century amelioration of a paraplegia by specific treatment was sufficient for the diagnosis of an exostosis reabsorbed by such treatment, also isolated cases of vertebral lesions were found at the post-mortem table. However, doubt began to be manifest among pathologists, and the opinion is now current that, although syphilitic lesions of the spinal column exist,—for there is no reason why the vertebræ should not be involved as well as any bony part of the body,—still such lesions are rare and are not always easy to determine, especially caries, as being of syphilitic nature. You will notice, in the *résumé* of the cases published as yet, that the anatomical verification fails in a certain number. Often a deformation of the vertebral column existed, and had some value for the diagnosis; but where this sign was wanting, one was obliged to depend on the influence of the treatment, which, however, is not always reliable. There is in reality a very limited number of cases in which, together with the spinal paraplegia, alterations of the vertebræ, probably syphilitic, have been found.

MENINGO-MEDULLARY SYPHILIS.

The massive gummata of the meninges and cord with considerable thickening of the meninges are rare occurrences. This does not mean that gummatous formations are to be considered exceptional; on the contrary, they are frequent, and are in a way one of the characteristics; but the circumscribed infiltration is usually reduced to a microscopic nodule. Like-

wise the thickening of the meninges is frequent, but it is generally not very pronounced, and seldom produces the condition which has been described by the name of syphilitic pachymeningitis.

When the insufficiency of the theory of exostosis was recognized, the prominent alterations of the cord and its membranes attracted attention. Although a certain importance was granted to syphilis in acute and chronic diffuse myelitis, the gummatous tumors of the cord and the gummatous and fibrous thickening of the meninges were alone considered to be characteristic.

The gumma is a common manifestation of syphilis, and there is no reason why such lesions should not appear in the connective tissue of the envelopes of the cord. Such observations are rare for macroscopic gummata. Transitional forms exist also between the slight diffuse gummatous thickening of the meninges and the massive gummatous tumor. It is necessary to say that the macroscopic appearances by which the syphilitic nature of the tumors is diagnosed do not always offer absolute certainty. Even competent histologists are often puzzled.

It is only with considerable reserve, and after a careful microscopic examination of the different parts of the spinal axis at regions where the elementary lesions are found, that one can admit the syphilitic nature of such tumors.

Together with the infiltrations, circumscribed or more or less diffuse, considerable fibrous thickening of the meninges is described.

Such alteration of the meninges, and especially of the dura, may be secondary to the osseous lesions of the vertebral canal which produce an external pachymeningitis. But the point of origin can be in the meninges themselves, and especially in the softer vascular pia. More rarely the lesion occupies primarily the internal face of the dura. Later all the envelopes are involved, become united, and form a thickening, which surrounds the cord sometimes, as a sort of fibrous collar.

It is remarkable that the meninges of the superior part of the cord are those usually involved in these cases, and that the dura plays an important part. The development of the process seems to follow the law of Jürgens. This author believed that the syphilitic lesions of the cerebro-spinal axis follow a descending march, and are propagated from the brain to the cord. In these cases, in reality, the lesions of the cerebral meninges, especially at the base, are very marked, and they decrease as more inferior regions of the cord are examined. But this law is rigorously applicable only to special cases, which can be classified under the designation of syphilitic cerebro-spinal meningitis, as proposed by Lamy.[1] Considerable thickening of the membranes and the participation of the dura can, however, be found in the regions of the cord at some distance from the brain.

The lesions of the nervous parenchyma are important, for it is to them

[1] Lamy, Thèse de Paris, 1893, p. 56.

that sooner or later are due the symptoms and often the death of the patient; but it is relatively only late that they acquire this importance in certain forms of meningitis, when the process has already attained an advanced degree. It is evident that in these cases the meningitic lesion can attain considerable intensity without at first producing grave lesions of the cord. This is a remarkable circumstance, which does not occur in the ordinary cases of medullary syphilis, as we shall see. It would seem to prove that it is not so much the considerable thickening of the meninges which has an influence on the condition of the medullary parenchyma as the alteration of certain important elements of the nourishing membrane,—of the vessels, especially. In the majority of cases of spinal syphilis, one has to do with more diffuse lesions than the gumma and the pachymeningitis, and it is by a histological study that one discovers the alterations of the cord and meninges. Numerous writers have spoken of the whitish and dull appearance of the pia and of the clouded condition of the arachnoid. Knorre has mentioned the existence of milky patches on the pia, which he regarded as capable of causing the thickenings and adherences of the membranes, or as being the point of origin of the gummatous developments. There does not exist in reality a clearly-defined distinction between the extended diffuse lesions, which are only appreciable by the microscope, and the macroscopic alterations represented by a considerable thickening of the meninges or the production of massive gummata. The different appearances are due to the duration of the affection. There are transitional cases where, by the side of macroscopic lesions, considerable microscopic changes are noted. Sometimes the cord appears normal to the naked eye; but it is possible by the exterior aspect, the consistency, and the coloration of the different parts, to recognize any important alteration in this delicate tissue.

The anatomical modifications of the syphilitic paraplegia with a rapid evolution can be properly appreciated only with the microscope. The cases of rapid progress are best fitted for the study of the initial lesions. In the prolonged cases it is possible to interpret them by the forms of transition, and to discover the connection of the different alterations.

In the gumma and the circumscribed thickening of the meninges, the cord is only secondarily involved, and relatively late. It is more common to find the meninges and cord equally involved in the syphilitic process, and marching hand in hand. To this condition has been given the name of syphilitic meningo-myelitis.

Julliard has attempted to show the predilection for the lymphatic system. This conception arose from the diffusion of the inflammation in the meninges and the perivascular sheaths. Gilbert and Lyon[1] have proposed the following classification:

1. The hyperæmic and necrobiotic meningo-myelitis, characterized by a congestion and perhaps a multiplication of the vessels of the cord and its

[1] A. Gilbert et G. Lyon, De la Syphilis médullaire précoce, Arch. de Méd., 1889.

coverings, which cause nutritive disturbance in the nervous axis, and finally the softening of the cord.

2. In the embryonic meningo-myelitis, cellular hyperplasia, diapedesis, and vascular exudations appear, and microscopically one finds a luxuriant proliferation of young cells in the vascular walls and in the substance of the pia and its intramedullary prolongations, likewise the production of a subpial fibrino-leucocytic deposit.

3. The disseminated round cells in the cord and meninges pursue their course, and end in the formation of an adult tissue, the meninges become thickened and united, the cord becomes hard, and the microscope shows the substitution of the meningo-medullary elements by a fibrous tissue, principally developed about the vessels, of which the walls undergo notable change in this diffuse sclerotic meningo-myelitis, as in the preceding forms.

4. Finally, the round cells accumulate at certain points in the form of small tumors, and undergo the degeneration proper to nodullary syphilitic formations, and thus is formed the gummatous meningitis, the gummatous myelitis, or the gummatous meningo-myelitis. According to this description, the alterations of the pia and cord are simply the result of an inflammation developed under the influence of the syphilitic poison, and the different forms correspond to variations in the evolution or duration of the same process.

We consider the alterations of the nervous parenchyma in the more common cases as primarily of a degenerative order, and as provoked by the troubles of nutrition resulting from lesions of the nourishing organs of the cord. This is not a new idea, but sufficient importance has not been paid to it.

Lamy [1] has recently stated that the primary lesions occupy the meninges and vessels, and that the nervous elements are involved secondarily, either by the direct propagation of the infiltration of the meninges to the cord, or by the circulatory disturbances which result from the vascular obliterations. These two forms of alteration usually coexist, with predominance of one or the other, and at times the changes of the meninges may be so slight that one would be almost justified in describing separately the phlebitis and arteritis of syphilitic origin.

We will go further. The specific infiltration can in certain cases be propagated from the pia to the neighboring nervous parenchyma, it can develop primarily in the cord about the vessels; but the infiltrations are usually very limited, except when they assume certain special forms (gummata, pachymeningitis). The important alteration is the necrobiotic change in the nervous tissue. Although secondary, this anæmic necrosis is, nevertheless, the principal lesion, and it is to this that is due usually the syphilitic paraplegia which we associate with a transverse myelitis.

In the forms with a rapid evolution, we may by the study of lesions of

[1] Lamy, loc. cit., p. 31.

recent origin establish the true nature of the process; but in the chronic forms the interpretation is more difficult. In the observations which we give, the variation in the form and diffusion of the lesions is a striking feature; however, the modifications in the meninges and vessels are constant, and Julliard has made of this localization the characteristic feature of syphilis. In the ignorance in which we are at present regarding the nature of the specific agent, we are not able to propose another criterion. While recognizing that the participation of the lymphatic and vascular passages, the diffusion of the lesions, and the impossibility of their systematization are the characters of the syphilitic inflammation, it is necessary to confess that there is nothing special in the process, and that the connective tissue and the lympho-vascular system are a common soil for the evolution of every infectious agent.

According to the general opinion the vascular lesions and those of the meninges are primary, the alteration of the nervous tissue is secondary; and there are two special forms of this alteration,—the softening of the nervous tissue with destruction of the noble elements, and the induration with sclerosis. The state of softening is much more frequent, in the cases of rapid progress it exists almost alone, and in those of chronic development terminated by an abrupt attack the softening is found together with more ancient lesions.

The softening of the cord in syphilitic myelopathy has been noted for a long time. It is sometimes accompanied by very evident macroscopic lesions of the meninges, but usually the meningitic lesions are observed only with the microscope.

The softening may be located at any point of the cord and may occupy an extent more or less considerable. It is sometimes localized at the periphery, sometimes in a central part, or it may involve a whole segment and assume a transverse type.

The intensity of the softening varies in degree. There may be complete destruction of the nervous tissue and the formation of a creamy substance. The process consists in a destruction more or less advanced of the noble elements with the formation of the products of necrosis, amorphous, granular, albuminous substance, granules of myelin, granular bodies, etc., and in a special alteration of the interstitial tissue peculiar to the nervous parenchyma. There is an exaggerated development of Deiter's cells. These modifications are not special to syphilis. In these cases there does not exist a trace of inflammation of the interstitial tissue, which proves that the destruction of the noble elements is not consecutive to an inflammation of the interstitial tissues, and that it is necessary to seek another explanation for this phenomenon.

In cases of syphilitic paraplegia without material lesions, their absence is probably due to the brief duration of the trouble and to the slight involvement of the meninges.

Recent works have called attention to transitory nervous symptoms

which occur in the secondary period,—headache, faintness, modifications of the pulse, etc.,—and which indicate the involvement of the entire nervous system. Perhaps in certain cases they may be exaggerated and cause particular forms of the paraplegia, but these manifestations ought to be separated from the common syphilitic myelopathy.

Writers speak also of the induration of the cord from sclerosis, and they agree in recognizing that if the softening characterizes the rapid forms, the sclerosis pertains to the chronic forms. The sclerosis is only then the last stage of the process,—of the inflammation of the interstitial tissue of the cord.

We would call attention to the fact that the tissue of the sclerosis is not always of the same nature. Sometimes—and this is not so common—it is a cicatricial tissue resulting from the organization of a specific infiltration. The gummatous infiltration arising from the pia or the vessels is characterized at first by an embryonic proliferation which can invade more or less the medullary parenchyma, and produce later a sclerotic tissue. The sclerosis gives a special appearance, the neuroglia does not enter into its constitution.

Usually the sclerosis is purely of the neuroglia; it is preceded by an inflammatory reaction of its own tissue, but this irritation is consecutive to the destruction of the noble elements. In cases of rapid evolution it is possible to constitute the destruction of the nervous elements at a time when the neuroglia does not present a trace of reaction. It is this neuroglia sclerosis which forms the zones of degeneration in the cord. Sometimes it is diffuse and seems then to be the result of a previous state of softening; sometimes it assumes a systematic disposition, and produces the secondary degeneration of the columns of white matter. Usually the spinal roots are not much involved in syphilis; however, they are at times sufficiently so to play an important part in the symptoms, and this is especially true in the very marked lesions of the meninges. Although many writers had spoken of the part played by the arteries in cerebral syphilis, it was due to Heubner[1] that a great advance was made in this direction. According to his view these lesions of the arteries may exist sometimes alone and at other times with syphilomata. The alteration is believed to arise in the internal coat between the fenestrated membrane and the endothelium, and consists in an accumulation of round cells. It would thus have an origin purely endarteritic and be produced by the irritation of the syphilitic blood; later it extends to the adventitia. While the proliferation of the intima is due to proliferation of the cells of the endothelium, the round cells which infiltrate the external coat come from the capillaries of this tunic, and again it is the direct action of the blood according to Heubner. This hypothesis has been contested. Credit has always to be given to Heubner for showing the frequency of arterial lesions in cerebral syphilis.

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[1] Heubner, Die luetische Erkr. der Hirnarterien, etc., Leipzig, 1874.

Baumgarten believes that the origin is in the vasa vasorum and the perivascular space of the adventitia, and that the proliferation of the internal membrane is secondary (Virchow's Archiv, vol. lxxxvi. p. 219). The modification of the circulation and of the nutrition which results from this change in the external coat provokes in the intima a lesion which can be propagated to portions of the vessel where the adventitia is sound. According to this conception the blood in the lumen of the vessels has not a great influence on the nutrition of the vessel: the nutritive current in the capillaries, and therefore the order of involvement, is from without inward. Lancereaux[1] acknowledged the same external origin, and explained it by the predilection of the syphilitic virus for the lymphatic tissues and the existence of the lymphatic sheath of His. Köster[2] believes that the primary lesion is a mesarteritis; that the origin is in the vasa vasorum, but the inflammation is first manifested in the muscular coat; and that the other lesions are due to a progressive invasion. Wherever the origin may be, the result on the circulation is the same.

As a point of distinction from the atheroma there is no calcareous and fatty degeneration; there are some exceptional cases which do not obey this law. These lesions which we have just described are applicable to the vessels of the whole nervous axis, to the cord as well as to the brain.

Greiff[3] seems to have been the first author who insisted on the alteration of the veins, and since his publication the change in the veins has been acknowledged as constant, even preponderant. The infiltration from the beginning involves the entire venous wall, and this thickening leads to contraction or complete obliteration of the lumen. The same writer remarked that the infiltration of the pia is always much more marked in the region of the vessels, and it appears that it extends from this vascular origin, and that the meningitis and the affection of the vessels have a close union in origin. At other points there is not this relation of the lesion of the meninges to that of the vessels, and the two processes have a certain independence.

Cases exist in which the vascular alterations are almost completely isolated, as in the case of Schmauss.[4] Here the inflammation was limited to the vessels, and even the smaller ones were involved, while the pia was sound. The arteritis of syphilis is a panarteritis, it involves all the coats of the vessels, and the hyaline change may lead to complete closure of the vessel. Möller[5] likewise has recorded a case where the lesion was confined to the vessels. In these two cases the paraplegia occurred abruptly, and death was speedy. There is a close resemblance in this to the cerebral

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[1] Lancereaux, Traité d'Anat. pathol., 1878–1881, vol. ii.

[2] Köster, Sitzungsb. d. niederrhein Gesellschaft für Natur. u. Heilk. in Bonn, 1874, and Berlin. klin. Wochenschrift, 1876, p. 454.

[3] Greiff, Arch. f. Psych., 1882, Bd. xii.

[4] Schmauss, Deutsches Archiv f. klin. Med., vol. xliv. p. 244.

[5] Möller, Arch. f. Derm. u. Syph., 1891, vol. xiii.

softening of syphilis by arteritis. Even in forms less pure the vascular lesions play an important *rôle*. In seven cases which we publish in this work, the vascular lesions were predominant, and we ascribe to them the principal alterations of the nervous tissue and the chief symptoms of the clinical picture. In short, in every form of syphilitic myelopathy the vascular alterations have been noted, and they are very early in point of origin. The modifications of the nourishing membrane of the cord are the first in date, and from these arise the changes of the cord proper. We attribute the softening to the circulatory disturbance due to the vascular lesions.

The physiologists have attempted by ligature of the aorta or by injection into the femoral artery of inert powder to produce an arrest of the spinal circulation. As a result of the various experiments we have two facts of importance: the production of a paraplegia and the softening of the cord; other symptoms are anæsthesia of the parts involved, paralysis of the sphincters, and grave trophic troubles. It has also been shown by the experiments that the circulatory alterations in the cord are capable of producing very marked subjective sensations. The result of the cord anæmia is the necrosis of the nervous elements. The gray substance is first affected. The cells undergo the process of degeneration, the fibrilla of the gray matter becomes varicose, the white matter is soon affected, the axis cylinder swells, the myelin becomes granular, the capillaries dilate, small hemorrhages take place, the cellular elements are increased in number, and many granular bodies appear. Later, the interstitial tissue reacts and produces the sclerosis. It is thus easy to study the evolution of the cord softening in the animal, and there are a few cases of paralysis of the lower limbs following obliteration of the aorta; however, in these there were symptoms due to peripheral arrest of the circulation. Just as all the lesions of the brain were regarded as inflammatory before the idea of softening was accepted, so it is certain that a large number of the inflammatory conditions of the cord which have been described were only ischæmic softening.

Vulpian [1] ascribed to the ischæmia of the cord certain clinical symptoms, and compared them to the attacks of cerebral origin. Such spinal paralysis is much more rare than the cerebral, due to the condition of the circulation in the cord. The anastomosis here is extensive, and the anæmia exists only in very extended vascular lesions. The circulation of the cord is not as well known as that of the brain.

In the medullary softening of compression myelitis, the necrobiosis is explained by the external pressure, and the term myelitis is not justified, for it is a degenerative process.

It is very probable that other infectious maladies may affect the vessels of the cord, and certain paralyses in the course or at the termination of infectious diseases, as typhoid, small-pox, etc., and which present the picture

[1] Vulpian, Maladies de la Moelle, 1879.

described by Landry, may be due to vascular origin. In spite of the vascular lesions of medullary syphilis it is only recently that an important *rôle* has been attributed to them in the lesions of the nervous tissue, and it is Lancereaux who has especially insisted on this process and only from the clinical phenomena. These consist in premonitory signs preceding the paraplegia, which is of abrupt development, and characterized by a loss of reflexes, participation of the sphincters, and trophic troubles; later, if the patient does not succumb, the sensibility returns gradually and the contracture and the other spastic phenomena appear in the inferior members. The abruptness in the appearance of the paralysis is a characteristic phenomenon of the medullary softening of ischæmic origin; it is seen in those cases where the vascular lesions were almost exclusive and in all grave cases of syphilitic paraplegia, but it occurs also in forms of moderate intensity.

The chief objection to the vascular theory is in regard to the distinction which is made between the cord and brain circulations, and to the anastomoses of the cord. These anastomoses only exist for vessels of a certain order; they can also explain a certain number of phenomena, as the collateral congestion and the capillary hemorrhages which accompany the medullary softening. The small vessels are, however, end arteries, and their injury naturally affects the corresponding parts of the medullary parenchyma. The cord receives its blood-supply especially by the arteries of the anterior and posterior roots, branches of the spinal vessels from the vertebral, intercostal, lumbar, and sacral arteries. These branches perforate the dura, and divide into an ascending and descending branch, which anastomose with the corresponding arteries, and thus are formed arterial chains of which the principal are: an anterior supply situated in the anterior fissure, and formed from the arteries of the anterior roots (tractus arteriosus anterior); then there are two symmetrical posterior chains (tractus arteriosi postero-laterales) situated between the posterior roots and the corresponding lateral columns. From these systems a net-work in the pia is formed, and from this net-work the arterioles and capillaries penetrate into the substance of the cord. Once detached from the pia these small vessels do not anastomose.

The gray substance and the white substance constitute two separate vascular departments.

The gray substance is irrigated by the central arteries, which arise from the anterior chain, and, according to some writers, there is an anastomosis near the central canal. At different points of the cord these branches penetrate into the gray commissure at the base of the anterior fissure, and, having attained one or the other side of the central canal, divide into ascending and descending branches, which anastomose with similar ones. The artery at each part of the cord goes only to one side of the central canal,—sometimes to the one, sometimes to the other side.

The white substance is furnished by arterioles from the whole periphery. The anterior columns and the anterior part of the lateral columns are trib-

utaries of the anterior arterial chain, while the posterior column and the posterior part of the lateral columns, comprising the pyramidal tract, are under the control of the postero-lateral arteries. According to Kadyi, there is a zone of white substance surrounding the gray, which is supplied by both systems. We have observed that the nervous tissue of this zone was always more respected, even when the peripheral and central parts of the cord were very much altered.

It is necessary to examine the condition of these peripheral end arteries; often they are much more altered than are the vessels from which they arise. The vascular systems may be involved separately, or at least the lesions can predominate in one, and thus give rise to the appearance of systematization, which the medullary syphilis often assumes; in this way can be explained the poliomyelitis anterior of rapid evolution in a syphilitic subject. We would expect the vascular changes to predominate in the anterior system in such a case.

The veins of the cord anastomose freely everywhere. The largest of these are situated at the posterior and anterior median fissures. In the syphilis of the cord the veins are usually more affected than the arteries, and these lesions, in spite of the anastomosis, have an important bearing.

The mode of *début* of the paralytic phenomena is different according to the case, and there are intermediate forms between the apoplectic and the slow paraplegia. The abruptness is considered as an indication of softening of anæmic origin. The vascular alterations can attain a certain degree of development, and only produce slight circulatory trouble; but at a given moment, from one cause or another, the equilibrium is disturbed, and the consequences of the vascular trouble are rapidly manifested. In examining the cases followed by anatomical verification which have begun abruptly, it is seen that this phenomenon has existed when the lesions were almost or entirely vascular. This rapidity is almost characteristic of the grave forms, for it indicates that many vessels are involved and in rapid succession, and yet the same thing occurs in mild forms.

The *début* can be apoplectiform, usually it is less sudden; it is after some days that the paraplegia attains its maximum. This abrupt beginning has been noted in about half the cases, and we believe with Dejerine that it is more common than is usually supposed.

The predominance of the symptoms in one side of the body, the fluctuations in the evolution, the relapses, the attacks of paraplegia more or less grave, finally the "fragmentation" of the symptoms, are all explained by the vascular lesions. The circulatory disturbance can affect the respiratory and cardiac centres, and cause sudden death. At other times death is from decubitus and trophic troubles.

We have slighted the alterations of the meninges, because these lesions are of secondary interest, and usually act on the nervous tissue by the vascular changes accompanying them. The process at first is localized here about the vessels.

In some cases the infiltration may involve the cord considerably, the inflammation of the meninges can cause thickening and adherence of its envelopes, or a gummatous neoplasm is developed. Such lesions joined to the vascular disturbance give a special seal to the syphilitic process, but they rarely acquire any importance in secondary lesions of the medullary tissue.

The compression from a pachymeningitis or gumma can also have an influence on the cord.

We have examined microscopically seven cords. In the first case a man of thirty-four years, syphilitic since two years, was afflicted with lumbar pains, heaviness in the legs, and trouble in the sphincters. Some weeks later he was seized by an abrupt attack of paraplegia, and died sixty hours afterward.

This is an important case in regard to recognition of the nature of the lesions at the beginning of the affection. The vascular lesions were very pronounced, the veins were especially involved, and this condition existed at all points, especially at the level of the bulb. There were some peri-vascular infiltration and small gummatous nodules in the meninges and cord.

The vascular alterations had their maximum of intensity in the superior dorsal region, and here the cord was softened for a long extent. There was not a trace of inflammation of the interstitial tissue. It was, in short, an ischæmic softening.

In the second case, a woman of thirty-four years had had syphilis since the age of twenty-six. Seven years after infection she presented the phenomena of diffuse cerebro-spinal trouble, which lasted some months, and was interrupted by an abrupt attack of paraplegia. After this the paralytic symptoms were accentuated progressively and rapidly, and the patient succumbed about five months after the attack of paraplegia. At the autopsy considerable vascular lesions and a marked infiltration of the meninges were found, with predominance of the inflammation about the vessels. At some points the gummatous infiltration developed in the walls of the veins formed quite large nodules, of which the centres were in process of cheesy change. The degeneration of the nervous tissue was more marked than in the first case. At certain points the invasion of the nervous substance by the specific infiltration from the pia or an intra-medullary vessel was noticed; but these lesions were much less extensive than the alterations of a degenerative nature. The alterations of the roots, which are very marked in this case, seem, on the contrary, to arise from a progressive invasion of the infiltration.

The third case presented anatomical lesions similar to the second, but as the duration had been longer there were noted the signs of repair, neoformation of capillaries in the walls of the vessels and in the thrombus, and reaction of the neuroglia.

The fourth case showed sclerosis of the neuroglia.

In all these cases the vascular alteration was constant, and the changes in the meninges were secondary.

FIG. 1.

Gummatous tumor developed in the wall of a large vein of the pia at the level of the bulb. The central part of the tumor is in a condition of caseous degeneration; the deep layers of the venous wall are affected by the same process. The lumen of the vessel is much flattened out, emptied of blood, and contains only some granular detritus spread along the internal surface. The peripheric layers of the neoplasm are formed by living cells, which invade the pia (in the right portion of the figure).

FIG. 2.

Arteries and veins in the neighborhood of the bulb. The arteries free in the subarachnoid cavity present the lesions of periendarteritis. A free vein is completely obliterated. The pia is relatively almost normal; it contains an inflamed artery, and a very dilated vein filled with blood.

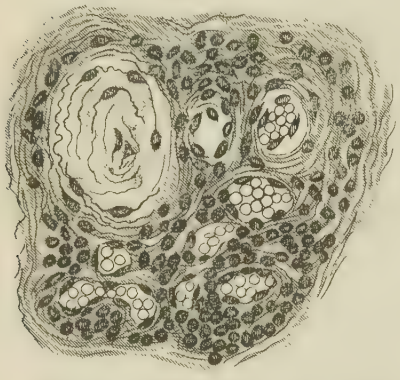
This fourth case shows that the alterations of the medullary tissue are due to the circulatory trouble, and not to invasion by a perivascular sclerosis, for in this case numerous altered vessels are found traversing sound

tissue, and the walls of the vessels which traverse sclerotic tissue have no adherence with it.

In the majority of the cases which permit the survival of the patient, the alteration of the medullary substance is situated in the dorsal region, the lumbar centres are preserved, but their function is modified by the presence of the lesions situated above and by the sclerosis of the pyramidal tracts. The patients present a spastic paraplegia. This is the type described by Erb.

The clinical picture depends on the location of the medullary lesions. These can predominate in the gray substance, the posterior horns, be distributed in disseminated foci, and thus form the pseudo-systematic forms, recalling tabes, the anterior poliomyelitis, the multiple sclerosis, etc.

Fig. 3.



Vessel of the white substance. Obliteration of the vascular trunk and compensatory development of capillaries in a nodule of infiltration of the external layer.

In the cases which we have examined, the lesions were usually more marked in the external coat of the vessels, and almost exclusively limited to this in the first stages of the process. In our cases it has seemed as though the middle coat and the elastic membrane offered a marked resistance to the infiltration, and it was only in cases of lesions very advanced that these were found involved. Small hemorrhagic foci are found in some places between the coats of the vessel's wall. We consider that there are two foci of origin for the vascular lesion,—from the intima and from the vasa vasorum,—for the syphilitic blood can act as well on the intima as on the endothelium of the nourishing capillaries; but we cannot agree with Heubner in thinking that usually the internal coat is the first affected,—we ascribe this priority to the external tunic, which is usually also the most involved by the lesion. However, the point of origin is of little importance, the endarteritis is the important lesion, since to this is due the contraction of the vessel, which can be the cause of thrombus formation.

In the cases which we have studied, where the affection was very recent the veins were more involved than the arteries. The infiltration begins in the exterior layer, but soon extends to the whole wall, which offers less resistance than the arterial wall. The vascular lesions are at first especially developed in the peripheral parts of the cord and in the pia, they involve also the free vessels in the subarachnoid cavity. The small peripheral end arteries of the cord take part in the process, and their lumina are often obliterated. The arterial or venous nature of the small altered vessels is very hard to recognize, but whatever may be the system to which they belong, the thinness of their walls favors the progress of the infiltration.

The infiltration of the vessels is usually diffuse, but there is a tendency to form miliary nodules, which sometimes surround a giant cell; and giant cells are found also in circumscribed infiltration not of a nodular appearance. These gummatous nodules are especially developed in the veins, and give a prominence to the exterior of the vessel; they can be of considerable size and may form veritable gummata. The miliary gummata are observed even in the cord; they develop always around a capillary. The vessels often become later solid cords, consisting of a hyaline substance. The small nourishing vessels of the cord are especially involved, and their early destruction explains the production of degenerative lesions in the medullary tissue at a time when the large arterial trunks are still permeable, and even very little altered.

The meninges, especially the pia, take considerable part in the inflammation. The predilection of the syphilitic virus for the lymphatic system ought to be put by the side of the irritant action of the syphilitic blood to explain the localization of the inflammation in the external layers of the vessels and the participation of the lymphatic spaces of the pia and of the arachnoid cavity. This participation is proved by the development of the infiltration between the pia and arachnoid; likewise an altered condition of the floor of the fourth ventricle is often constituted. We emphasize the fact that the infiltration of the pia is especially developed about the vessels. In following the progress of the inflammation it is noticed that it begins in the vascular walls and the adjacent lymphatic sheaths, afterwards it extends gradually, gains the lymphatic system of the pia, and from this moment develops in a manner to a certain degree independent. It is logical to admit that the syphilitic poison arising in the vessels has its first influence on these vessels, spreads then in the lymphatic sheaths, and from there in the entire spinal lymphatic system. In some cases it appears as though the lesions were primarily meningitic, when the vessel is as though choked, but the study of the early forms, and the intensity of alteration of vessels freed from all adherence in the arachnoid cavity, prove well the vascular origin of the inflammation.

The alterations of the nervous parenchyma following the changes of the vessels can be progressive or consist in a rapid necrosis. The diminution of consistency appears often rapidly, but not always. There is an abundant exudate of an albuminous liquid, and often small capillary hemorrhages. This exudate strangles the constituents of the cord. The vessels, especially the veins, are dilated and filled with blood. When the cord is cut at the autopsy, the white substance is found diffluent, badly separated from the gray, and the latter is often deformed. Even when the necrosis has the disposition of a transverse lesion, the transverse extent of the cord is not entirely destroyed: it is especially the marginal zone which is involved and only in second place the gray substance.

A very frequent form in the distribution of the zones of degeneration is that which affects the peripheral part of the cord in a long extent. This

results from the inflammation predominating in the peripheral vessels which supply nutrition to the marginal zone; that in the gray substance of the altered region is often less intense.

The secondary degeneration of the direct pyramidal tract we have seen extend as far as the first sacral roots. It is probable the secondary degeneration appears between the nineteenth and thirty-second day. The anæmia of the cord is never as great as that of the brain. The sclerosis is always secondary,—is never the primary lesion. To examine the details of this structure the cuts should be treated with acetic acid and potash, which swell the neuroglia, afterwards washed with alcohol and ether, which remove the fatty products of the disintegration. In the posterior column this sclerosis is always proportionally more dense.

Is it possible that the nervous tissue once destroyed is capable of regeneration? It is very evident without making intervene the regeneration of the elements destroyed that certain conditions, as the limitation of a process at first extended and of a diffuse nature, or the return of the circulation after momentary disturbance of the equilibrium in the parts not yet smitten with death, can explain the amelioration which one observes after the period of grave accidents is once passed. But one sees patients improve, after having presented during months and years the phenomena of intense spastic paraplegia with extreme contracture, producing even functional impotence, and at the end of a long period of very slow and progressive amelioration attain a state quite satisfactory. It is possible that the regeneration of the nerve-tubes plays an important *rôle* in this amelioration. Experiments on animals after section of the cord have shown reparation of the nervous substance and return of function; and we believe that for the nerve-tubes at least the regeneration is possible, for the nerve-cells the point is disputable. These elements once destroyed, no trophic power can intervene for their reproduction. The nerve-tubes are only conductors, the existence of which is subordinate to that of the trophic centres from which they emanate. In the cord as in the peripheral nerves they are capable of regenerating as long as this centre persists, provided that the cause of destruction which involves their course ceases to exist. In the medullary softening from syphilitic lesions of the vessels, the anæmia, however intense it may be at first, does not persist completely, and the vitality returns partly in the altered regions. The neuroglia sclerosis remains as an obstruction to the re-establishment of the normal structure, but this process is only secondary, it does not attack directly the nervous elements, it replaces them only after their destruction. It ceases to form when the degeneration is arrested. The dense sclerosis cannot be penetrated by the axis cylinders of new formation. In examining carefully the ancient cases of spinal syphilis small axis cylinders are found, isolated, clearly rounded, perfectly colored, and surrounded by very thin sheaths of myelin or else naked. It is hard to say if these represent new tubes in process of formation. We state only that an affirmative response

would accord with the clinical facts. "The clinic," says Leyden, "causes us to consider as probable the faculty of regeneration of the cord in man without proving it in a precise manner. Following wounds, hemorrhages, softenings, it is not rare to constitute gradual amelioration so striking that it is necessary to admit a partial regeneration."

We insist on the fact that if the infiltration of round cells about the vessels in the pia or cord have an extreme importance as characteristic of the process, they are only slightly developed, and often late; the principal lesion is the anæmic necrosis of the nervous parenchyma. The cicatrization of this infiltration takes the aspect of very dense connective tissue, often disposed as a wedge, with its base on the pia, with which tissue it is continuous.

There are lesions which directly affect the roots, produce direct symptoms, and cause secondary degeneration in the cord. For the anterior roots there is the secondary degeneration due to alteration of the cells in the anterior horns, but the radicular lesions are especially a direct consequence of the inflammation which invades the vascular, lymphatic, and connective tissues of the roots. Here one finds the point of departure of the infiltration in the walls of the vessels, it insinuates itself in the interfascicular connective-tissue spaces, and invades thus the body of the root. The destruction of the nerve-tubes here seems rather to result from the progressive invasion by the infiltration than from the circulatory troubles.

In the forms which terminate by rapid death when the infiltration is not yet much developed, and rests localized to the perivascular regions, the roots are little altered while the necrosis of the cord is intense. On the contrary, when a certain duration of the affection has permitted the extension of the inflammation, the perineurium is thickened and infiltrated, the nerve-tubes are destroyed by the infiltration, which invades the body of the root. Later, when the lesions are cicatrized the root is composed of a sclerotic tissue which differs from the neuroglia sclerosis of the cord in the predominance of the connective tissue element. The alterations of the roots are usually little accentuated; however, in certain cases they assume a capital importance, and some authors have admitted a special form of neuritis and perineuritis.

SYMPTOMS.

As the lesions are multiple the symptoms are the same, but as the vascular changes have a certain fixity, there is a resemblance between the different clinical types corresponding to these lesions. Still there are variations in the common form due to more or less rapidity of the process, the portion and extent of cord affected, the presence of alterations in the brain, etc. The vascular changes can predominate in a certain system of vessels, and produce the symptoms of a systematic affection of the cord. By most writers the medullary syphilis of the ordinary type is divided into acute and chronic, which correspond almost to the terms grave and benign. Intensity and rapidity are usually associated characteristics, but not in-

separable. Usually it is the great extent of the vascular lesions which determines in the cord the changes at the same time rapid and extensive; still a lesion developed rapidly may be circumscribed, and the symptoms after a stormy beginning may moderate and yield to a benign form. Likewise the progressive alterations may invade extensive regions, and the case then becomes more acute.

The acute form is associated with flaccid paralysis, muscular atrophy, and grave trophic troubles; on the other hand, the spastic phenomena characterize the chronic forms.

Vinache[1] describes the first symptoms as a sense of torpor or heaviness, irradiated pains in the legs and body, and genito-urinary difficulty. He speaks of vacillating march, the "fragmentation" of symptoms, and of the predominance of motor troubles.

According to Hammond,[2] there is a slow form, beginning with pains irradiated from the vertebral region towards the extremities, and characterized by a muscular stiffness of gradual development. Then occurs the paraplegia, beginning first in one leg, later involving both. Sensation is only slightly affected; the sphincters do not act normally. Anæsthesia and trophic disturbance appear late. The recovery is slow. But the paralysis may appear rapidly, and be the first symptom excepting some vague indications.

A transitory circulatory trouble can be the origin of a flaccid paralysis, which does not persist if the anæmia has not existed long enough to cause a necrosis of the nervous tissue. This is the same as the paralysis of cerebral origin from cerebral arteritis. The symptoms, though never absolutely unilateral, can predominate in one side.

The rapid form begins often with pain, and the paralytic phenomena appear quickly, the motor and sensory troubles soon attain their maximum, the sphincters participate in the paralysis (first retention, then incontinence), the reflexes are abolished, and decubitus appears.

Erb has sketched a form in which the gait is that of spastic paraplegia, the reflexes are exaggerated, but the contracture is moderate, the bladder is always involved, sensation is only slightly altered. There are no severe pains, muscular atrophy fails, arms, head, cranial nerves are free. The affection is progressive. There exist paræsthesia, temporary pains, lassitude, weakness, and stiffness of the legs. The spastic paresis may be great, rarely is there complete paralysis. It is remarkable that the contracture is so slight. The tendency is to amelioration, especially with energetic treatment, and improvement is found in about half the cases. Erb likewise speaks of a rapid evolution in some cases and paraplegia in a few days. We believe this simply indicates a more rapid extension, and we believe also that the constancy of Erb's form is due to the constancy of the location

[1] Vinache, Thèse, Paris, 1880.

[2] Hammond, A Treatise on the Diseases of the Nervous System, 1880.

of the lesion. The centres of the lumbar cord are not destroyed. If the transverse lesion is produced abruptly, it causes a flaccid paralysis, later a spastic form.

Erb's type is realized only when the lesion permits the survival of the patient for a sufficient period, and under certain other conditions.

We understand the clinical picture to be due to the vascular alterations primarily. The first symptoms of spinal syphilis are insidious, in other cases the *début* is rapid, or again it may be more gradual. Much oftener we find after a period of diffuse symptoms an abrupt aggravation in form of an attack of paraplegia, which either persists and is soon followed by death, or else there is improvement with spastic symptoms. It is not necessary for this spastic period to be preceded by an attack of paraplegia. In the premonitory period there are diffuse circulatory troubles; the meninges may be involved and likewise the roots. Frequently there are cerebral symptoms, for cerebrum and cord are one, and are equally exposed to the syphilitic blood, or rather the cerebrum is more so. In the beginning cerebral symptoms are usually present, such as early headache, temporary ocular troubles, paralysis, etc. These, with spinal symptoms, have diagnostic value; they do not indicate a descent from brain to cord, as Jürgens believes, but simply that the brain was first involved. There is a certain opposition between brain and cord, a persistent paralysis from cerebral softening is not followed immediately by spinal paralysis of the same intensity. The alteration oscillates on the cerebro-spinal axis, but once fixed it exhausts itself at this point.

PERIOD OF PREMONITORY SYMPTOMS.

The usual premonitory cerebral symptoms are more or less violent headache, vertigo, vomiting, diplopia, with temporary ocular paralysis, possibly trouble of vision, sometimes violent delirium, loss of consciousness, coma, intellectual disturbance. The principal character of these lesions is diffusion and fluctuation. For spinal symptoms there are often first the disturbances in the sensation, pains in the back, loins, and limbs, and paræsthesia. Pains in the loins may be slight, but increased by every movement, even that of the head. Others suffer more when lying down. It is exceptional to find the pains atrocious, though they are often more intense during the night, as seen in the nocturnal cephalalgia. The location in the loins, or the dorsal region between the shoulders, is more common than in the cervical or sacral regions. The pain may be present only on pressure, or it may exist in the form of a girdle, or be less severe in the lower extremities than in the back, for shooting pains are exceptional. Usually there are tingling, numbness, sensation of warmth or cold, and little or no loss of objective sensibility at first. The motor disturbances are usually in the lower extremities, and may consist in rigidity, especially after repose, feebleness, increased reflexes,—these, however, may be decreased or vary from day to day. The sphincter troubles are often early and of diagnostic value,

in connection with light and fluctuating disturbance of motion and sensation. They may be the first symptoms. While usually insidious and progressive in development they can at times be rapid or sudden, being in such cases usually preceded by disturbance in motion or sensation, but this is not always true, and then such unannounced sphincter difficulty is generally followed by rapid paralysis of the extremities. Before the condition of impossibility to urinate there is usually the waiting, the pressing, the "hesitation." Frequently there is a slight retention, with impulse to repeated micturition. Then there are the troubles of the genital system. That which is characteristic of the premonitory period is the extreme fluctuation of the symptoms and their diffusion, due to the circulatory lesions. Dejerine relates a case of syphilitic claudication,—inability to walk after going a short distance. There is only one symptom which offers a certain permanence, and that is the pain in the back, which is due to inflammation of the meninges, and is found especially in the slow forms when the perivascular inflammation extends to neighboring parts. This is usually absent or slight, in the rapid form, because the anæmic necrosis of the nervous tissue comes before the involvement of the meninges. This premonitory period is variable in time, rarely lasts many years, usually is of a duration reckoned by weeks, and may be interspersed with periods of considerable improvement.

PERIOD OF ORGANIC CHANGES IN THE CORD PARENCHYMA.

A time arrives when the nervous tissue is no longer altered functionally but organically; this may be gradual or acute in development. Ordinarily the premonitory symptoms become gradually worse, and one day after some sudden exertion the legs of the patient refuse to sustain him, he has time to support himself by a table or to drag himself to a bench, often after some moments he is able to rise, or in other cases it is only after some days his legs resume their function. This accident may repeat itself several times before permanent paralysis occurs, and may be associated with transitory retention of urine. These transitory paralyses are usually in the daytime. It is a kind of syncope of the cord, which being badly nourished is not equal to exertion. Nocturnal accidents are more grave and more permanent.

The permanent attacks come in the same manner as the premonitory, the patient rarely falls as though struck by lightning, rarely is there loss of consciousness; usually the recollection of the event is perfect. The paralysis often comes during the night, the patient awakens paralyzed in the legs, and there has been no warning during sleep; the same is seen in cerebral paralysis. Sometimes the paraplegia may require several days for its development, and then usually begins in one limb alone, and may be limited to this for several days. The clinical picture of the mode of beginning is often varied. When the paralysis is once established there remains usually the power of movement of the toes and of slow flexion of the

leg, with inability to raise the heel. At this time the paralysis is flaccid; this does not mean a destruction of centres, but is due to decreased circulation. To the same cause are due the muscular twitchings, which indicate irritation of reflex centres. It is seldom that reflex activity is so great as to cause at once permanent contracture, but at the end of some weeks in bed the legs are found in extension or demiflexion with great rigidity. Often the muscles of the lower part of the trunk are affected in this paralysis, and the patient is unable to sit up in bed, or to maintain himself thus when he has been so placed. If the paralysis remains flaccid, the paralyzed muscles lose their tonus and become soft.

The objective sensation is always altered at the moment when the paraplegia begins, and there may be complete anæsthesia in the affected parts, or it may be moderate or even partial,—*i.e.*, affecting certain qualities; but disturbance of sensation is always less than that of motion. There may be hyperæsthesia: the slightest touch of the skin may be painful, and this in close proximity to zones where pinching and pricking are hardly perceived. There may be paræsthesia, and one of our cases, at the moment of commencement of the paraplegia, felt a blow on the calves of the legs as if from a whip. The severity of the pain in the back is not in proportion to the gravity of the paralysis. The troubles of the sphincters are never absent in the permanent paralysis. The three great characteristics of the clinical picture at the *début* are the symptoms affecting motion, sensation, and the sphincters. At the beginning of the paralysis there is ordinarily no marked reaction, no fever, and the organic functions (except those of the sphincters) are as usual; sometimes the pulse is full, and there may be shivering, vertigo, vomiting, general malaise, and sensation of choking.

Death sometimes occurs in the first day of the attack, due to the extent of the lesion in the cord or to involvement of vital centres, and then we find signs of acute bulbar paralysis, precipitate and interrupted respiration, cardiac accidents, hiccough, and death occurs before decubitus develops; but this rapid termination is exceptional, and usually there is a period of longer duration. If the paraplegia is light, amelioration may set in early, and the patient enter almost at once into the condition of spastic paraplegia.

In the grave forms decubitus soon develops, and if death does not occur in the first days (this event is exceptional), the symptoms persist, and either the reflexes remain lost or rigidity occurs. The troubles of sensation seldom remain as grave as at first, it is rather an attenuation for all forms, or sometimes one quality is principally involved,—touch, pain, temperature, etc. The persistence of anæsthesia is a grave sign, likewise the aggravation of this, for such anæsthesia manifests itself only in cases of great destruction of an extended segment of the cord. There is atrophy of the paralyzed muscles to a certain measure, due to destruction of the motor cells, and this is accompanied by reaction of degeneration. Idio-muscular contractions are at first exaggerated, but, as the atrophy increases, they weaken or disappear. The atrophy is especially in the lower extremities, but may be as

great in the lower part of the trunk; it is, however, often hidden here by adipose tissue, or else it is considered simple emaciation. It would seem that the destruction of the gray substance of the dorsal region has less severe consequences in point of view of the nutrition of the muscles than has destruction of the cervical or lumbar centres. The decubitus has been noted in regions free from pressure, and often trophic lesions have also been described. Death, after the paralysis, is usually from marasmus, or purulent infection, or other complication. At times there is improvement, even after symptoms of intense degree, instead of death. The absence of marked atrophy and the presence of contractures, even if only moderate, are good signs, for they indicate that the lumbar cord has not been destroyed.

PERIOD OF SPASTIC PARAPLEGIA.

In the period of spastic paraplegia the spinal centres for the sphincters, like those for movement of the lower limbs, are cut off from cerebral impulse, but react to peripheral, and so a patient may not be able to extend the limbs, which are rigid in demiflexion, but is able to accomplish this movement by scratching vigorously with the nails the skin of the anterior surface of the thigh, while another patient performs urination by pinching the skin of the abdomen. Obstinate constipation may yield to diarrhœa and fæcal incontinence for a time. Muscular contractions may occur spontaneously in the lower limbs, and cause the patient to jump in his bed, and sometimes awaken him at night; they may occur from the least irritation, as the jarring of his bed. Sometimes there are veritable crises of contractions: the limbs, moderately stiff, extend or flex slowly without the control of the patient, his attempts to prevent them only increasing them. Although the anæsthesia may have been complete at the beginning, it is never so at this period, for by this time it has become modified. Usually there is a perversion of sensation, a hyperæsthesia, perhaps only for certain forms of irritation.

Lumbar pains are rarely noticed in the period of spastic paraplegia; usually it is a numbness or coldness in the legs which is complained of. The patient may be confined to his bed for years, with the upper extremities, the cranial nerves, and the intelligence normal, but usually this intense paralysis is absent. As a rule, the voluntary movements are free from incoördination, but are performed as if the patient worked by springs. Sometimes the muscular rigidity is slight as long as the person remains in bed, but the latent contracture shows when he gets up, and when sitting it may be difficult to separate the knees, and one leg can be crossed over the other only with the aid of the hands. This is not weakness, as is proved when he resists communicated movement. The rigidity in the act of walking may become so great that the hips are fixed by the contractions, and the gait is like that of a duck. There are forms where certain symptoms of tabes are joined to the spastic paraplegia.

The evolution occurs as follows: Some months or a few years after the

syphilitic infection the patient experiences the premonitory symptoms, and later a paralytic attack, which may attain its maximum in some hours; or if this attack aborts the previous symptoms become more severe, and he is confined to his bed. In moderate forms, at the end of from four to ten months the grave symptoms have disappeared, and the patient has entered on the stage of spastic paraplegia.

In some cases of superacute form the prodromic period fails, or passes unnoticed, and the patient is stricken with an apoplectic attack, and death follows after ten days or two months. In other cases there is a slow progression to spastic paraplegia, uninterrupted by accidents.

There are forms which never go beyond the premonitory stage. When the stationary period has been attained relapses are not common. Death is common in the acute forms.

The persistence of the chronic state of spastic paraplegia is due to sclerosis of the cord; therefore recovery is rare if the disease has progressed beyond the vascular stage.

There are some cases of superacute *début* which begin in a most grave form, ameliorate for some days or weeks, and even end in complete recovery. The only explanation is, that a large portion of the cord has been involved in the vascular lesion at one time without the anæmia having been sufficient to destroy the vitality. This is equally true of certain forms of hemiplegia of cerebral origin, and they could be called functional.

The acute forms are those in which the greater part of the cord is involved; the chronic those in which the lesion is transverse and with the lumbar swelling free. Although the lesion is principally in the white substance, the gray matter is never intact. When the modifications of tissue at the seat of trouble are slight and disseminated, the sclerosis of the column of Goll and of the lateral columns presents the appearance of primary degeneration of these tracts,—*i.e.*, of a combined sclerosis. The primary lesions, although scattered, are sufficient to destroy many nerve-tubes.

The location of the trouble in the lumbar cord produces a flaccid paralysis, atrophy, anæsthesia, etc., and differs from the ordinary location in the severity of the symptoms and in early death.

PSEUDO-SYSTEMATIC FORMS.

Syphilis may produce systematic myelitis, the anatomical process of which is different from the myelopathy which we have been studying; clinically there is also a difference between the true systematic affections and those which result from the predominance of diffuse lesions in certain systems of the spinal axis. Ordinary tabes may be more or less faithfully simulated; likewise muscular atrophy, contracture, and tremor; the cephalic phenomena may, by their predominance or association, recall anterior poliomyelitis, multiple sclerosis, lateral sclerosis, etc. The tabetic form may follow an attack of paraplegia, or may develop progressively, and closely resemble true tabes; still there are usually certain atypical features.

The form of combined tabes is the most common, in which spastic and tabetic elements are united. The pseudo-tabes can be distinguished from the true by the mode of *début* and evolution, which are more rapid, by the frequency of acute accidents, and by reaction to specific treatment.

Sometimes the syphilitic lesions appear almost entirely limited to the lateral columns, and the degeneration is in the direct cerebellar and the pyramidal tracts, simulating a primary lateral sclerosis; but on the whole this form is so slightly distinguished clinically, and by distribution of the lesions, from the ordinary form that it hardly merits to be considered apart.

Muscular atrophy is common in the grave forms, especially in the period which follows the attack of paraplegia, and foretells the persistence of grave symptoms, for it announces the destruction of motor cells. In the forms which terminate in death, the atrophy is usually pronounced if it has had time to develop; on the other hand, in the forms which terminate in spastic paraplegia this symptom often fails. In some cases the muscular atrophy presents a special form of the disease; and yet there are usually certain features which distinguish it from true poliomyelitis, as lumbar and peripheral pains, spastic phenomena, trouble of the sphincters. The question arises whether syphilis can act directly on the motor cells as it is believed to act on the posterior columns in tabes, and be one of the causes of progressive poliomyelitis. Siemerling believes that the alteration of the cells results from hemorrhage, softening, or round-cell infiltration, and we must express our view as favoring the opinion that it is of secondary origin, and probably in most cases due to alteration of the nourishing vessels of the gray substance. The vascular lesions of the gray horns are the chief disturbance in these cases, the alteration of the medullary tissue elsewhere, although it exists, being, by reason of its minor importance, of secondary rank. This form may develop more or less rapidly, and thus produce the acute or chronic poliomyelitis.

In certain cases the symptoms can be grouped, and assume then the form of bulbar paralysis. This is only a question of location, not of difference in the lesions.

The gummata of the cord do not cause any sign especially characteristic; as an exclusive lesion it probably produces the symptoms of slow compression. It would seem that it ought to cause the appearance of demi-section of the cord, but clinical observation does not support this. One is inclined to make such a diagnosis in the clinical picture of a hemilesion in a syphilitic person, especially if amelioration is obtained by specific treatment; but in the case reported by Charcot and Gombault, which presented this form in all its purity, the autopsy revealed the existence of unilateral lesion, but not a gumma. Gummatous or fibrous thickening may occur at any region of the cord, and sometimes this involves the cauda equina, and the cord above escapes.

Fournier has described a syphilitic neurasthenia, consisting in intense headache, trouble of sensation, hyperæsthesia, anæsthesia, neuralgic pains, a

little everywhere, weak legs, sometimes exaggerated reflexes; but there are no organic signs, no vesical nor oculo-pupillary troubles; it resists specific treatment, is especially diurnal, and sometimes true hysterical signs are present.

While the abrupt *début* of the paraplegia is characteristic to a certain point, especially if preceded by vacillating symptoms, still such paraplegia does not necessarily mean softening in the cord, for a lesion near the cord, of slow development, may cause this, as shown in a case observed by Dejerine, where a syphilitic patient was afflicted with rapid paraplegia, muscular atrophy, vesical and rectal trouble, decubitus, and at the autopsy a sarcoma was found on the vertebral column, and compressing the cord.

Hysterical paraplegia can give a picture very hard to diagnose from the syphilitic form; the same is true of certain cases of multiple sclerosis. One must likewise be on his guard in diagnosing a polyneuritis as a syphilitic spinal atrophy, but usually spastic signs fail, and the sphincters are not involved. The syphilis may be spastic, with muscular atrophy and dissociation in the sensation, and thus closely resemble syringomyelia; but the rapid and oscillating march and the coexistence of other symptoms indicate a diffuse lesion.

As for prognosis, if the lesion is confined to the vessels and meninges, the specific treatment can accomplish much; later it has little action on the lesion already produced, but can prevent further development. The treatment should be vigorous at the first appearance of involvement of nerve tissue, three to six grammes of mercurial ointment daily, with hygienic precautions; likewise four to six grammes daily of the iodide of potassium.

If the symptoms develop slowly there is more hope of arresting the process by treatment. The prognosis is more grave when the location is cervical, and more so when lumbar than when dorsal.

Although the acute onset is very dangerous, there may even then be almost complete recovery. The signs of danger are bulbar involvement, respiratory trouble, paralysis of the diaphragm, grave trophic trouble, purulent cystitis with albuminuria, and especially hectic fever. Under such circumstances the mercury should be stopped and the iodide of potassium alone given, but if the primary symptoms return it is well to resume the mixed treatment.

As to the frequency of syphilis of the cord opinions differ: usually it is considered a rare location. Among one thousand and eighty-five cases of syphilis of the nervous system recorded by Fournier, only seventy-seven cases were purely spinal, and four hundred and sixteen cerebro-spinal. Erb believes syphilitic paraplegia to be ten times less frequent than tabes. This paraplegia is sometimes very early, sometimes late, more frequently it is near the period of infection. There is no relation between the severity of the infection and the spinal location; the weight of opinion is towards a previous mild manifestation, and our own opinion, derived from the cases we have observed, agrees with this.

The neuropathic heredity does not seem to play an important *rôle* in the spinal location. The exaggeration of the vulnerability of nervous tissue by hereditary predisposition favors the development of parenchymatous affections, but this etiological factor does not seem to enter into count when it has to do with inflammatory lesions involving interstitial tissue. The heredity is shown in another manner,—by the transmission of the specific germ; and although nervous affections are rare manifestations of hereditary syphilis, they can be found in the cord. The male sex furnishes the greater number of cases of syphilitic paraplegia, and the same is true of every paraplegia, not because of the rarity of syphilis in females, but because of the excesses, overwork, etc., which are more common in the male sex. It is more common before middle age, for, as it appears soon after the infection, it is natural that it should occur at a period when infection is most common.

Rheumatism seems to be an important predisposing cause, according to some authors, likewise overwork, cold, traumatism; but we are not disposed to ascribe the vascular and meningitic location to these occasional conditions, although when the primary lesions are once established they may have important influence. In the precarious state in which the circulation of the cord is, the least excess becomes overwork for a tissue where nutrition is already deficient.

We would form our conclusions as follows:

1. Syphilis can act on the nervous system in two ways:

First, directly; in attacking the parenchyma, it determines thus at the *début* of the affection the first vague nervous troubles of the secondary period, and later, perhaps, certain systematic affections, as tabes. This mode of action is not clearly explained, for there are no anatomical characteristics which permit us to recognize the origin of the affections which are attributed to it.

Second, indirectly, in producing an inflammation of the vascular, lymphatic, and connective-tissue elements. The alteration of the parenchyma is secondary to these lesions. The reality of this process cannot be disputed, it is affirmed by the aspect of the inflammatory lesions, which, although not special to syphilis, are, nevertheless, to a certain point characteristic of this affection. The process can strike all parts of the cerebro-spinal system, but is limited sometimes exclusively to the cord.

2. Syphilis of the cord appears at a period near that of infection, with a maximum between the end of the first year and the end of the sixth, and is much more frequent in men.

3. The inflammation begins with the vascular walls and perivascular regions and involves especially the small vessels of the periphery of the cord. In the large vessels it involves the internal, and especially the external tunic, developing about the vasa vasorum. From this point it involves the perivascular lymph-space, afterwards the lymphatic system of the meninges, and finally the entire arachnoid cavity. The infection spreads by the circulatory system, and rapidly in the lymphatic system, where it assumes an independent form. At this period the lesions are constituted by :

An inflammation of the vascular walls, which attains its maximum in the veins and small vessels.

A diffuse general infiltration of the connective tissue of the meninges.

An irritation of all the surfaces bathed by the cerebro-spinal fluid (surfaces of the meninges, ventricular walls).

These inflammatory lesions are characterized by a tendency to nodular formations (miliary gummata of the meninges, of the vessels, of the cord).

4. The alterations of the nervous parenchyma, the essential elements, and of the neuroglia are secondary; they may result from imperfect nutrition, on account of the vascular lesions of the cord and of the nourishing membrane, or from an invasion of the medullary parenchyma by the specific infiltration. The first is the more important cause.

5. According to the intensity, the distribution, and the rapidity of evolution of the primary lesions, the anæmic necrosis of the nervous tissue appears abruptly, as a transverse softening, which may be located at different points of the cord or predominate in one or the other vascular department; or else it appears slowly, and then the destruction is accompanied by a process of substitutive reaction of the neuroglia, which replaces the destroyed elements. This period of substitution is favored by the partial return of the circulation (collateral circulation, development of the vasa vasorum, formation of new capillaries in the obliterated vessels), and terminates in the neuroglia sclerosis. The connective tissue which enters the cord with the vessels is also thickened.

6. Although the necrobiotic lesions followed by sclerosis constitute the principal alteration, there are certain medullary and especially radicular changes, which result from the invasion of the nervous tissue by an infiltration extending from a point in the meninges or a perivascular sheath. This process can in certain cases assume a considerable importance.

7. While the lesions preserve the same characters, they may vary in their distribution. They are generally diffuse, but they sometimes assume the aspect of a transverse lesion, more or less intense, more or less limited, and located at different heights of the cord.

They can be distributed more irregularly in a considerable extent of the cord. In every case they are more marked in the marginal zone. The dorsal location is the most frequent.

Be the lesions confluent or be they disseminated, the result is always the same, and they produce the effect of a transverse lesion, accompanied by a secondary degeneration ascending and descending. The lesions involve especially the territory of the postero-lateral spinal vascular system. They may predominate in certain regions of the cord,—the lateral columns, the posterior columns, the gray substance of the anterior horns,—and thus simulate certain systematic affections.

8. The ordinary clinical evolution is the following: At the period of formation of the primary vascular lesions and of those of the meninges, there are diffuse premonitory phenomena.

At the period of softening and of degeneration of the nervous elements, there is an attack of paraplegia, followed by paralytic phenomena and grave trophic troubles.

At the period of sclerosis there is the chronic spastic paraplegia.

The abrupt *début* can be manifested without being preceded by a prodromic phase, or in other cases the spastic paraplegia comes slowly without passing through the acute stage.

9. Death may occur either in the first period of the affection from the localization or extent of the lesions, or more slowly from the progress of the affection or from a complication.

The ordinary termination of the affection is a spastic paraplegia persisting in a chronic state after an amelioration more or less marked.

The complete recovery is only possible in certain conditions, when the primary vascular and meningitic lesions have been arrested before the final destruction of the nervous parenchyma.

The reorganization of the necrosed nervous tissue, if it is possible, is manifested only in a limited degree.

10. In certain conditions the primary inflammation is accentuated in the meninges, producing a meningitis or a pachymeningitis, or else it assumes the form of a circumscribed gummatous neoplasm.

11. The iodo-mercurial treatment is demanded at the appearance of the first symptoms. It acts only on the primary inflammatory productions, and is without influence on the necrobiotic lesions once established.

12. The medullary syphilis is always a serious affection. Death may intervene in spite of treatment, especially in the acute forms. Outside of certain rare fortunate cases where complete recovery is obtained, the amelioration never goes beyond a certain limit, which is fixed on account of an incurable sclerotic cicatrice of the cord.

SOME OBSERVATIONS WHICH APPEAR TO ESTABLISH THE AERIAL TRANSPORTATION OF MALARIAL GERMS.[1]

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An interesting paper read before this society last year by Dr. Wm. H. Daly, of Pittsburg, setting forth the fact that malarial diseases may be and, in Dr. Daly's opinion, ordinarily are disseminated by drinking water con-

[1] Read before the American Climatological Association at Hot Springs, Va., June 13, 1895.

taining the germs of the disease, has aroused some discussion and has arrested the attention of a number of medical men.

The controversy as to whether malaria comes from the air we breathe or the water we drink is by no means new. In fact, it is as old as medicine itself. According to Major Smart,[1] Hippocrates believed that the manifestations of malarial diseases were due to the use of marshy waters; but Galen and Avicenna recognized the existence of a marsh-poison which contaminated the air. In 1831, Orton,[2] in his work on cholera, instanced the unenlightened state of the Hindoo mind, inasmuch as they believed that malarious diseases came from bad drinking water. Sir Joseph Fayrer[3] also mentions this belief of the natives of India in his celebrated Croonian lectures of 1882, and adds that they also believe that the milk of buffaloes or cows pastured in places overflowed by waters containing the malarial poison has the same effect in producing the disease as the waters themselves. As we are not informed of the dairy methods of the Hindoos, we cannot judge whether the milk may be contaminated by water subsequently to milking or not. We infer, however, that it may become noxious in this way.

The case of the soldiers on the transport "Argo," in the year 1834, recorded by M. Boudin,[4] is familiar to most of you, and clearly shows that the cause of the outbreak of malarial fever upon the vessel was the marsh water, which those affected drank, and which those who escaped the fever did not drink. And so strong was the chain of evidence in this case that M. Colin,[4] who was convinced that malarial infection was solely conveyed by the air, was obliged to dispute the diagnosis and assert that the fevers were cases of typhus,—a quite unwarranted assumption.

Many observers, especially Sir J. Fayrer,[3] Surgeon E. G. Russell[1] (in his work on "Malaria and Diseases of the Spleen," Calcutta, 1880), Hehir,[5] Sternberg,[6] Smart,[7] Parkes,[4] Moore[4] ("Diseases of India," London, 1861), agree that the malarial poison may enter the system with the drinking water. Dr. Smart[7] refers to the great improvement in the health of the garrison at Fort Brown, Texas, especially in the matter of malarial diseases, since a better water-supply has been obtained and the troops no longer drink the Rio Grande River water. Dr. M. K. Taylor[4] made similar statements with respect to Fort Sill, Indian Territory, in a paper read before the International Medical Congress at Washington some years ago. Dr. Richard Waggoner,[7] of Pensacola, Fla., gives a number of instances which show that the malarial diseases in his neighborhood depend upon the drinking water. Cases like those of Dr. Jones,[6] recently reported, of Drs. Norburg,[6] Lewis,[7] Daly,[6] Lumsden,[6] Boyker,[6] etc., show undoubted contamination of the drinking water and the propagation of malarious disease by this means.

Professor Bemis,[23] of New Orleans, has shown that the poison of malaria may live in the water an indefinite and undetermined time, and be conveyed by water currents through immense distances and even to remote islands

and continents. Smart [4] tells us that the disappearance of malarial disease from a locality has evidently been often associated with the substitution of percolated water for surface supplies. The Terrai, a belt of marshy country lying along the base of the Himalaya Mountains, has recently been reclaimed and made habitable by the use of deep wells. The testimony shows that in this tract the chief cause of the malarial fevers lay in the surface waters which had been used as a drinking supply. Parkes [4] says, "One very important circumstance is the rapidity of development of malarious disease and its fatality when introduced in water. It is the same thing as in the case of diarrhœa and dysentery. Either the fever-making cause must be in larger quantity in the water or, what is equally probable, must be more readily taken up into the circulation and carried to the spleen than when the cause enters by the lungs."

Dr. von Wedekind,[6] U. S. Navy, states that it is a common practice for medical officers serving on the West African coast to forbid entirely the use of the "land water" as a beverage by the men under their charge, which might seem to some a reversion to the enlightened state of mind noticed in the Hindoos in 1831.

Hertz [8] says that in colder climates, Germany, the Netherlands, Russia, etc., malarial fevers most commonly appear at the time of the melting snow. Dr. Smart [9] showed that the malarious diseases affecting the troops at Fort Bridger, Wyo., increased in direct ratio with the organic impurities of the drinking water, and were especially prevalent when melted snow-water found its way into the stream from which the drinking water was derived.

My friend, Dr. Atkins, of East Las Vegas, New Mexico, noted, in the spring of 1894, a violent dust-storm from Deming, New Mexico, to the Raton Mountains. He says, "After the storm had raged all day in the Rio Grande Valley it was curious to see at 5 P.M. the very fine dust sailing high over the mountains west and southwest of Las Vegas coming from all New Mexico southwest of us. In such a way our water might have been infected."

The doctor had been suffering with a severe chill and fever, contracted, as he fancied, from the drinking water. He says further, "In Las Vegas and vicinity we have absolutely no malaria, no low or marshy places, but at Albuquerque and other Rio Grande points it exists." He also adds that the seasons of 1893 and 1894 had been unusually wet, after six or seven years of heat and drought, and in the fall of 1894 a few cases of what was apparently malarial fever and of typhoid fever, the first in three or four years, were noted. His conjecture of the aerial contamination of the water-supply of Las Vegas, which comes from a pond on the side of the mountain, over which the dust-storm blew, is reasonable and probably correct, although the pond water used for drinking might have been contaminated by the heavy rains. Rains after droughts are universally prone to cause malaria whether swamps or bogs are near by or not, and they probably do

this by sweeping organic matters into the water-supply. It is said by some, however, that these rains wash the malarial germs out of the air into the water.

Smart[4] also mentions the fact that malarial hæmaturia, hemorrhagic malarial fever, etc., are prevalent in winter when atmospheric malarial exhalations could not be expected, and states that remittents and so-called typho-malarial fevers may occur when the temperature is below zero, and therefore must be supposed to come from the water.

Those of us whose practice lies in a growing community, where a public water-supply has been recently introduced, have all, I presume, noticed cases of disease of a malarious nature, the victims of which were still drinking from the "best well in town." Without referring to my case-books, I can recall a number of instances in which "malaria" disappeared from a household just as soon as they began drinking the "town water" and give up the ancestral well. Dr. Whitehorne, of Verona, New Jersey, called attention some years ago to the increased freedom from malarial diseases enjoyed by the inmates of the City Home, a correctional institution of Newark, New Jersey, since they had ceased drinking surface water. Dr. Harvey, in the same discussion, stated that, in his experience, malaria comes from the drinking water. Whether my cases above mentioned were true malarial diseases or not, I, for one, am somewhat doubtful. It is a very fortunate thing that the fact of the existence of the plasmodium in every case of true malarial poisoning seems to be proved. And it is to be hoped that the recent reports that each of the commoner forms of malarial fever has its distinct plasmodium will be verified. With this aid to diagnosis, the nature of many an obscure affection will probably be made manifest, and intelligent direction will be furnished for the action of health officers and sanitarians in the matter of water-supplies, drainage, etc.

The writer agrees with Dr. Daly,[6] so far as this, that the text-books do not as a rule sufficiently emphasize the fact of malarial infection from drinking water. It is, perhaps, needless for me to remind you, however, that the probability of the aqueous spread of malaria is mentioned in Reynolds's "System of Medicine" (Dr. Maclean), Hare's "Therapeutics" (Dr. Dock), Pepper's "American Text-Book" (Professor Whittaker), Ziemssen's "Cyclopædia" (Hertz), Loomis's "Practical Medicine," Bristowe's "Theory and Practice of Medicine," Pepper's "System of Medicine" (Ed. 1884), Niemeyer's "Text-Book of Practical Medicine," "The Medical and Surgical History of the War of the Rebellion," and, turning to the writings of that noble clinician, Dr. George B. Wood, we read ("Practice of Medicine," part i. chap. ii.), "It is not impossible that they [the malarial miasmata] may be absorbed through the skin and even through the mucous membrane of the stomach, which they may enter with the saliva."

The writer remembers instances during his army service in New Mexico in which the consumption of watermelons seemed to be provocative of

malarial disease. I only mention this in passing, because Dr. Daly lays so much stress upon the chance of malarial poison being taken into the system by eating spinach, lettuce, and other fresh vegetables which may have been washed in marshy waters and may have the germs adhering to their surfaces. The possibility of the communication of malaria by the latter means or by the juices of fruits or melons grown in malarial soils is a question requiring further investigation. Dr. Cadwallader[6] states that drinking the sap of the sugar-maple and eating sorghum molasses are probably sources of malarial fevers, and Hertz and others warn travellers against eating watery fruits while in malarious countries. Dr. Daly, in the paper referred to, seems to lay great stress upon the fact that he and his comrades were able to remain in the swamps for considerable periods without contracting ague, so long as they did not drink the marsh water. And the inference which he seems to wish to be drawn from this fact is that the air of the swamps does not contain malaria, and that, therefore, there is no aerial transmission of malaria. There is one fallacy in the doctor's position which he seems to have overlooked. The comparative immunity of those dwelling in swamps from chills and fever has been a matter of comment, and, I may say, of surprise, for a number of years. At any rate, Sir Thomas Watson noted, in his admirable lectures, the fact that among the inhabitants of the Dismal Swamp, malaria is never seen. Atkins[11] says, "It must also be admitted, however, that these diseases do not prevail in all marshy districts." Dr. Geo. H. Horn[12] reports an absence of malarial disease at Camp Independence, California, where every supposed factor for its propagation existed. Osler[13] says that it is impossible to tell from the soil whether it is malarious or not. Sternberg[19] said, in 1883, that there is still a factor in the development of malaria with which we are unacquainted and whose precise action we can only surmise.

Smart says that owing to local causes the vicinity of a marsh may not be an insalubrious locality, and in another place says that a swamp or marsh is not needed for the development of malarial diseases.

Singapore is a place whose freedom from malaria can be explained only by assuming that there is not sufficient change either daily or annually in the temperature to produce the radiation of heat necessary to set the malarial germ in motion.

Dr. Baker (Hare's "System of Therapeutics," vol. i. p. 536) tells us that "in many countries, through draining the soil, clearing of forests, *and other measures tending to reduce the difference between day and night atmospheric temperature*, there has been a very great reduction in the sickness from intermittent fevers."

The Amazon River, and many marshy localities in Australia, New Zealand, New Caledonia, South America, and the Bermudas are mentioned as free from malaria, where the contrary would be expected.

Rusby[6] observed that in travelling in South America there is little danger in sleeping near stagnant pools, but that a sojourn near running

streams or water-falls was almost sure to be followed by a seizure. The editor of the *Medical Record*, in commenting upon this observation, says, "This would seem to argue in support of the theory advanced some time ago that the malarial poison is carried by running water from wooded regions, and that marshes are not its natural habitat."

Hertz[8] says, "Instances occur every now and then in which, with every condition present for the development of malaria, this poison is entirely lacking." We cannot account for these exceptions unless it be on the ground of the disinfecting properties of ozone, which is said to be largely developed in some marshes.

If, then, the atmosphere of certain marshes is free from malaria, may not Dr. Daly and his friends have been hunting in such a marsh, and hence escaped the miasma, as soon as they desisted from drinking the water?

Having now considered the question of aqueous transmission of malaria, we turn to that of its aerial transmission. As hinted in the early part of this paper, belief in the latter proposition has always existed.

Pliny[1] noted the fact that trees destroy or consume mephitic vapors, and Galen and Avicenna recognized the existence of aerial contamination with marsh miasmata. Lancisi[10] in 1695 published his "De Noxiis Paludum Effluviis," in which he proved the aerial origin of malarious disease, and this work was for years the great authority upon the subject, and still has an effect upon the medical mind.

Tomasi Crudeli,[3] than whom probably no man living has made a more extended study of the natural history of malaria, showed a number of years ago that a perpendicular elevation of fifteen or twenty feet will stop the malarial current, but an object of the same height with inclined or sloping sides is not efficacious in arresting the passage of malaria. The same authority[15] also points out that people sleeping on platforms three or four metres from the surface of the ground are comparatively safe from malarial disease. He also directs that the air of the morning and early evening hours be rigidly excluded from dwellings, especially if any excavation may be going on in the neighborhood. If possible, air to ventilate the house should not be taken from near the ground, but should come from a higher stratum. Flowers should also be rigidly excluded from houses in malarious regions.

Dr. W. O. Daniel[16] recommended keeping the doors and windows shut as much as possible between sunrise and sunset, and states that excellent results have followed the execution of this plan.

Dr. John M. Ward,[6] superintendent of the New Jersey State Lunatic Asylum, observed no case of malarial fever among six hundred and seventy-two inmates of the institution, all of whom were kept in-doors after sunset, whereas the laborers and assistants in the asylum, who were not so restricted, suffered with such diseases. Hartshorne[20] noted that the in-door operatives in factories in miasmatic regions enjoyed comparative immunity from malarial disease.

Dr. Geo. B. Wood [17] stated that he was physician to a public institution containing more than one hundred inmates, among whom, during sickly seasons, autumnal fever was very prevalent until the order was enforced prohibiting any one from going out of doors before breakfast or after tea. We are told that screens or mosquito-nets will exclude malaria as well as insects.[3]

Stanley, in his well-known work "In Darkest Africa," says that Emin Pasha always took a mosquito-curtain with him, as he believed that it was an excellent protector against miasmatic exhalations of the night.

Stanley also says, "While ascending the Congo with the wind astern we were unusually exempted from ague. But descending the Upper Congo facing the wind, we were smitten with most severe forms of it; while ascending the Aruwini, we seldom thought of African fever, but descending it in canoes, meeting the wind currents and carried towards it by river flow and paddle, we were speedily made aware that acclimatization is slow." The Amazon [14] is also free from malaria, compared to its tributaries, which fact is explained by the assertion that a wind constantly blowing up the wide stream from the sea tends to equalize the day and night temperature and to obviate the nocturnal radiation of heat. Another explanation might be that the sea-breeze drives back or neutralizes the malarial germs contained in the land breeze. The Orinoco,[14] in South America, is a notoriously malarial stream and its great cataract is by far the worst portion. This cataract is surrounded by bare, black rocks and the radiation of heat is extreme. It is also true that the spray is dashed high and the malaria, originally in solution in the water, may be disseminated through the air by mechanical means as well as by the heat radiation. This view of the writer is confirmed by the opinion of Geo. B. Wood,[14] who says, "the atmosphere may be impregnated with malarial poison by the spray of water in motion."

We might observe here, in passing, that the view seems to be daily gaining ground that malarious waters are often made so by the washing down of the malarial germs of the air into the water. Combe's [18] experiments lend color to this view. He asserts, after four years' observations, that a light rain-fall lasting but an hour will purify the air completely of bacteria, and that after a snow-fall of a half hour the atmosphere remains completely freed from bacteria for a considerable time.

Smart [1] believes that rain-falls carry down the malarial germ from the air, and Geo. B. Wood and many others believe that malaria comes down with the dew.

It has been known since the time of Hippocrates that fires at night are an efficient means of keeping off malaria, their prophylactic action being due either to the prevention of the dew or to the creation of artificial currents of air and the intercepting of the established currents.

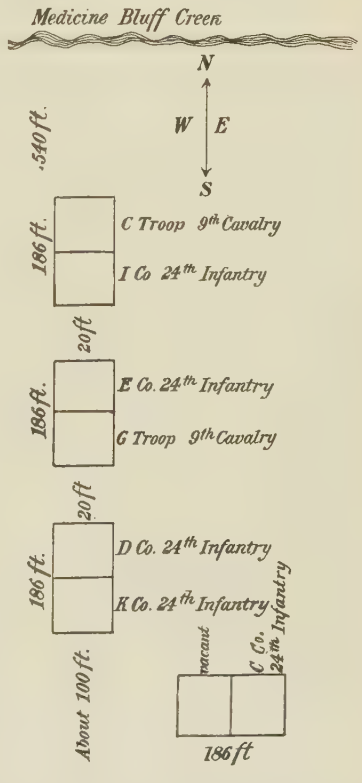
The fact of the carriage of malarial germs for long distances in the air is commonly believed by medical men living in malarial regions. Smart [1]

says, "Many medical men of the Mississippi Valley are of the opinion that during an especially unhealthy season the miasm from the river bottom affects persons living on the generally healthy uplands twenty or thirty miles inland from the eastern shore, but not on the western side. This is accounted for by the direction of the wind, which blows generally across the valley from the west."

Maccullock thought that malaria was blown entirely across the English Channel from Holland.

In another place Smart says that it is well known that the insalubrity of Sierra Leone is due to the winds from the Great Bullam Swamp. The windward side of most swamps is wholesome and the leeward may be if there be an obstacle to the air currents.

I do not believe it desirable to take up your time with further quotations tending to show the aerial transmission of malaria. But, if you will pardon me, I will add a few of my own observations to those already given. I will quote from a paper which I published in the *Medical News*, Philadelphia, of July 5, 1884. The writer became post-surgeon in this post (Fort Sill, Indian Territory) in the latter part of February, 1883, and was relieved from this duty November 20, 1883. During this period (about nine months) he attended "sick call" himself (with less than two dozen exceptions) and took entire charge of the cases in the hospital. He therefore is certain that all the cases diagnosticated as malarious received antiperiodic treatment, and believes that the diagnoses were correctly made. . . . Without doubt the hygiene of this fort is execrable, owing to the clayey soil and rank vegetation and to the disregard of every hygienic law evinced by the builders of the barracks. . . . All the barracks for the men are substantially similar. The accompanying diagram will show the disposition of these buildings as regards the creek, from which our water-supply comes, and the points of the compass. Each building is one hundred and eighty-six feet long and is divided equally by a transverse partition, each half accommodating one company of soldiers. The distance from the shore of the creek to the northern end of C Troop's, Ninth Cavalry, quarters is five hundred and forty feet. A passage-way of about twenty feet separates the three buildings, which run north and south from



each other. Nearly a hundred feet farther south is the barrack of C Company, Twenty-fourth Infantry. "Medicine Bluff Creek" is a stream of varying volume: the water has always more or less vegetable matter suspended in it. When the stream is swollen it widely overflows its banks, and subsiding deposits great quantities of dead leaves, grass, etc., in varying stages of decay along the banks, and even in the forks of trees and tops of bushes. The creek water is hauled about the post in a "water-wagon," and distributed for drinking and culinary purposes. It is kept in clean barrels and other receptacles.

The garrison of this fort is composed of six companies (until October 8th last there were seven) of colored troops. The prevailing wind is from the south. The next most frequent wind is the north wind, called here "Norther." It is always much colder than its opposite, and is popularly believed to bring more malaria. The direction of the winds at this post for the years 1880, 1881, and 1882, taken from the observations of the signal office here, are as follows: Three observations daily.

1880.		1881.		1882.	
Direction.	No. of Observations.	Direction.	No. of Observations.	Direction.	No. of Observations.
N	247	N	228	N	294
N.E	102	N.E	58	N.E	58
E	43	E	73	E	78
S.E	227	S.E	106	S.E	122
S	215	S	376	S	331
S.W	74	S.W	41	S.W	59
W	11	W	22	W	21
N.W	158	N.W	96	N.W	96
Calms	121	Calms	95	Calms	95

Very soon after beginning to attend "sick call" the preponderance of cases of sickness from certain companies excited my curiosity; and a little reflection led to the belief that the position of the various quarters, with reference to the creek, had some connection with the number of cases reported sick from that company. Accordingly, the following tables were prepared.

TABLE I.

SHOWING WHOLE NUMBER OF CASES OF SICKNESS FROM EACH COMPANY IN THE GARRISON FOR NINE MONTHS, AND WHOLE NUMBER OF DAYS OF SICKNESS.

	Cases.	Days.
C Troop, Ninth Cavalry	71	231
I Company, Twenty-fourth Infantry	48	168
C Company, Twenty-fourth Infantry	44	139
E Company, Twenty-fourth Infantry	39	115
K Company, Twenty-fourth Infantry	28	82

D Company, Twenty-fourth Infantry, left this post for another station October 8, 1883, and G Troop, Ninth Cavalry, was "in the field" for nearly four months. To make Table I. more complete we have

TABLE I. A.

FOR SEVEN MONTHS.

	Cases.	Days.
C Troop, Ninth Cavalry	51	152
I Company, Twenty-fourth Infantry	35	124
C Company, Twenty-fourth Infantry	32	100
D Company, Twenty-fourth Infantry	28	91

TABLE I. B.

FOR FIVE MONTHS.

	Cases.	Days.
C Troop, Ninth Cavalry	21	57
C Company, Twenty-fourth Infantry	17	44
E Company, Twenty-fourth Infantry	12	34
G Troop, Ninth Cavalry	12	29
K Company, Twenty-fourth Infantry	9	29
I Company, Twenty-fourth Infantry	8	22

TABLE II.

AVERAGE DURATION OF CASES.

	Days.
C Troop, Ninth Cavalry	3.25
I Company, Twenty-fourth Infantry	3.50 (or 3.02)
D Company, Twenty-fourth Infantry	3.25
C Company, Twenty-fourth Infantry	3.16
E Company, Twenty-fourth Infantry	2.94
K Company, Twenty-fourth Infantry	2.92
G Troop, Ninth Cavalry	2.16

A case of typho-malarial fever which terminated badly after six weeks in the hospital, and which was the only case of continued fever during the period, might, I think, be fairly left out in computing I Company's average sickness.

There are variations in the number of enlisted men in a company. The full strength of a troop of calvary is sixty-six, of a company of infantry fifty. The mean strength of any company varies constantly from enlistments, discharges, sickness, "detached service," etc. In Table III. the percentage of cases of sickness to the mean strength (*i.e.*, men actually on duty) is shown.

TABLE III.

PERCENTAGE OF MEN SICK WITH MALARIA IN NINE MONTHS.

C Troop, Ninth Cavalry	138 per cent.
I Company, Twenty-fourth Infantry	128 per cent.
C Company, Twenty-fourth Infantry	118 per cent.
E Company, Twenty-fourth Infantry	106 per cent.
K Company, Twenty-fourth Infantry	68 per cent.

TABLE III. A.

PERCENTAGE OF MEN SICK WITH MALARIA IN SEVEN MONTHS.

C Troop, Ninth Cavalry	98 per cent.
I Company, Twenty-fourth Infantry	94 per cent.
C Company, Twenty-fourth Infantry	89 per cent.
D Company, Twenty-fourth Infantry	71 per cent.

TABLE III. B.

PERCENTAGE FOR FIVE MONTHS.

C Troop, Ninth Cavalry	38 per cent.
I Troop, Ninth Cavalry	20 per cent.

TABLE III. C.

PERCENTAGE FOR ONE MONTH (September, 1883. The most sickly).

C Troop, Ninth Cavalry	46 per cent.
I Company, Twenty-fourth Infantry	34 per cent.
E Company, Twenty-fourth Infantry	30 per cent.
E Company, Twenty-fourth Infantry	20 per cent.

Mean strength of the command for the period, 277.

Total number of malarial cases 269, or 97 per cent.

Total number of days' service lost to the government by malarial sickness among the troops, 855.

Average duration of all cases, 3.02 days.

A comparison of the tables and the diagram will show that the number of cases of sickness and days of sickness increase in each company in direct ratio to its proximity to the banks of the creek, with the single exception of I Company, Twenty-fourth Infantry, which holds the third place in Table I., the fourth in Table II., and the third in Table III. Why? Obviously because the barracks of this company lie directly across the course of the north wind, and, in consequence, present a greater surface to the air currents than those buildings whose long axis is parallel with the direction of the prevailing winds (*i.e.*, north and south.) Were the south wind as unwholesome as the north, K Company, Twenty-fourth Infantry, would have the same average of sickness as C Troop, Ninth Cavalry, but not so great as C Company, Twenty-fourth Infantry (since the latter, as has just been explained, presents a greater surface to the prevailing wind). Leaving out of consideration C Company, Twenty-fourth Infantry, we observe the singular fact that there is a regular gradation in the number of cases in each company, per cent. of cases to mean strength, and days of sickness, as we recede from the creek.

If any one still adheres to the theory of malarial disease so ably advocated by Oldham,[22] he might say that the north wind being, as has been stated, so much colder than its opposite (see note) might bring malarial disease into the post by chilling the soldiers. To which the obvious reply is that it is inconceivable that a distance of one hundred feet farther north or south would make such a difference in the temperature of the wind as to have any effect upon those cases of sickness alleged to be caused by the wind.

I am aware that some remarkable instances have been recorded of the

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[NOTE.—Without being able to collect precise data, it is quite safe to say that the fall of temperature will exceed 20° F. within two hours of the setting in of a "Norther."]

growth of a line of shade-trees, or the removal a few hundred yards of a dwelling or barrack, stopping malarial fevers. But no observations of bodies of men alike in age and condition, eating the same food, drinking the same water, wearing similar raiment, and performing similar duties, have shown, so far as I know, that a difference of residence of less than one hundred feet from the banks of a stream will make a difference in the number and intensity of cases of sickness occurring on the same days. The banks of the creek afford all the supposed factors for the production of malaria, and to my mind the proof is satisfactory that the miasm was produced at the stream and brought into the post by the north wind. This miasm must, as has been pointed out by various observers, soon lose its potency in the air; or, what would have the same effect, may very soon be precipitated and fall to the ground, otherwise at Fort Sill, C Company, Twenty-fourth Infantry, would have the highest instead of the third place in the tables, since their exposure to the noxious wind is greater than that of any of the other companies.

The blacks seem about as liable to malarial poisoning as the whites in this garrison, although it should be stated that, owing to the small number of white men in and about the post, a fair comparison of the relative susceptibility of the Caucasian and negro races could scarcely be made.

The amenability of the b'acks to treatment was, however, quite marked, as the average duration (3.02 days) of the malarial attacks and the slight mortality (only one fatal case) among them shows.

In conclusion, then, I think we may say that both sides of the question —*i.e.*, the aerial and the aquatic transportation of malarial germs—have been proved. A controversy upon the subject would repeat the celebrated duel of the two knights over the bimetallic shield.

I think it fair to assert that in places ordinarily non-malarial the drinking water is probably the more frequent cause of ague, when it does appear, than the atmosphere, certain conditions, which we cannot always explain, but which are often, as in case of the water-supply of Las Vegas, mentioned above, quite explicable, impregnating the water. But in places like the Campagna of Rome, the Orinoco River in South America, and the jungles of East India, where malaria is so constant and so deadly, the atmosphere is the usual method of its transportation.

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CLINICAL LECTURES.

TUBERCULOUS PERITONITIS.

CLINICAL LECTURE DELIVERED AT THE MASSACHUSETTS GENERAL HOSPITAL, JANUARY, 1895.

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GENTLEMEN,—I am about to show you a patient who has been under observation, off and on, for a long time, a fact which adds much to the value of the case. My knowledge of him covers a period of upward of four years.

F. P. is now twenty-three years of age. He entered the hospital on the 25th of September, 1889. His family history was negative. A year before admission he had mumps, followed by orchitis, and a few months later swelling of the glands of the neck appeared, with suppuration following; he was troubled with bronchitis and hemorrhoids during the summer. Two weeks before admission he took a fresh cold and complained of soreness in the abdomen, more marked on the right side. At this time the abdomen was symmetrically enlarged, measuring thirty-four inches in circumference, and contained free fluid. There was fluid in the right pleural cavity up to the scapular angle. Shortly after he entered the hospital he was transferred from the surgical side to my service in the medical wards.

The right pleural effusion underwent absorption. On the 4th of Janu-

ary, 1890, he was discharged from the hospital with very little fluid in the belly, and no fluid in the chest. November 3, 1890, he returned with signs of a considerable collection of fluid, localized in the right side of the abdomen. He had, meanwhile, been perfectly well, save a slight attack of grippe, and the cervical glands had almost healed.

On the 8th of November, 1890, I tapped his belly in an unusual place. I tapped him exactly at McBurney's point, because we had evidence that the fluid was no longer free in the peritoneum, but was encapsulated in the right side. There is the little scar. I took out three quarts and a half of highly-colored, slightly bloody fluid, which had a specific gravity of 1017. The girth of the abdomen on entrance was thirty-one inches and a half; after tapping it was twenty-nine inches and a half. On the 20th of November he was discharged.

He reported again October 14, 1891. In the *interim* he had had caries of a rib, which healed up. October 4, 1892, he reported again, feeling well.

To-day the patient comes to us because he has recently "taken cold" and has a little cough, but he is feeling comparatively well. He has been working steadily and hard in a provision store in Cambridgeport, doing pretty long hours. Lately he has changed his occupation and is now on the road, getting more fresh air, and is altogether more favorably situated. The cough leads me, of course, to examine his lungs with great care; but I find nothing, and think he has simply a little bronchitis. To-day he feels first rate. Here you see the marks of suppuration. The glandular trouble was bilateral and fairly extensive, but has entirely healed. Here is the scar of the rib caries. He weighs now one hundred and forty-two pounds, as much as he ever weighed in his life. His belly looks perfectly natural. The bowels act perfectly well, there being no trouble with them. The temperature I took at my office, and it was normal. The liver edge I can feel distinctly, descending on full inspiration. The upper border of the liver is not raised, but the organ is evidently somewhat enlarged. The spleen is also enlarged; you will rarely have a better chance to feel a spleen. On deep inspiration the firm edge comes down against the finger. The increased size of these organs is probably hyperplastic. The general condition of the patient is too good to allow us to seriously entertain the idea of amyloid change.

Here is a man who is to-day practically well, and who has been well for four years, although during the year previous to those last four years he had peritoneal, probably pleural, glandular and osseous tuberculosis. Now this case teaches us at least two very important lessons. In the first place, it teaches a lesson as to the natural history and prognosis of peritoneal tuberculosis. But first allow me one word with reference to the diagnosis of tuberculosis. We have not, in this case, what you may call the clinching evidence,—the discovery of the bacillus. It was not found in the pus obtained from the glands, nor was it found in the fluid drawn from the peritoneal cavity. I think it was not looked for in these fluids; there was no

injection of the fluid into guinea-pigs. The bacteriological evidence is lacking. But for all that, I think there is no question as to the correctness of the diagnosis. What else besides tuberculosis could account for the various lesions observed in the case of this man,—suppurating glands, peritonitis, a carious rib, and pleural effusion?

Now look in Flint's "Practice of Medicine," in the edition of 1886, which was published, it is true, after the death of Dr. Flint, but which was edited by Dr. Welch, of Johns Hopkins University, and the present Dr. Flint. In this work it is stated, "The termination of tuberculous peritonitis is always fatal, either from the disease itself or from tuberculosis of the lungs or other organs. The duration is from one or two months to a year or more." I cite Flint's "Practice" as being a thoroughly representative book, a book which in its day was perhaps the best all-round book on the practice of medicine. Dr. Flint was a man of very large experience and a very careful observer. It may be added that this statement stands unchanged in the seventh edition (1894), edited by Dr. F. P. Henry. That statement, we will say, in the year 1886, represented the opinion of the profession in general,—that the prognosis of tuberculous peritonitis was necessarily fatal. We do not hold that view to-day. What has led us to change our opinion? Modern surgery has led us to change it. The first case that I recall is a case of Spencer Wells. He operated on a young woman with a large abdomen, supposing the enlargement to be due to an ovarian cyst. When he opened the belly he found a large collection of fluid, which was not encapsulated, and a peritoneum studded with bodies which, to the naked eye, resembled miliary tubercles. The fluid was drained out, the wound was sutured up, and, to his surprise, the patient got well. She married later and became the mother of a family. Then came other cases. Other operators had similar experiences, and it was not unnatural that the idea should gain ground that the curative agent, you may say, in these cases was the surgical operation, and that tuberculous peritonitis is a surgical rather than a medical disease. That view we cannot hold to-day; we have to recognize clinical facts, the most important of which is that tuberculous peritonitis gets well under simple general treatment without any interference whatever. I could adduce a number of instances in support of this statement. I will cite only one, the case of a man who, some three or four years ago, was taken acutely ill with symptoms which, for a week, left me in doubt as to the diagnosis between typhoid fever and peritonitis. Effusion then came on, showing that I had to deal with the latter condition. Soon the patient had an extension of the inflammation to both pleuræ, first one and then the other, with some effusion in the right and little or none in the left. Gradually the disease subsided, and, after lying in bed perhaps six weeks or two months, the patient went to the country. A few months later he took a position requiring close confinement, but has remained well ever since. The sceptic might say, "That was not a case of tuberculosis." I could not bring him any clinching evi-

dence; I cannot bring the bacillus; I cannot give a history of the guinea-pigs killed by the injection of the fluids. I could not convince a sceptic; but, at the same time, his scepticism would not affect my opinion. I believe this was a case of tuberculosis. I see no other way of explaining the simultaneous inflammation in the peritoneum and pleuræ in the absence of rheumatism or other obvious septic infection. So let us accept one clinical fact,—that tuberculous peritonitis may get well spontaneously under simple medical treatment. Our patient is an illustration of the fact that the disease gets well after tapping. He had it for upward of a year before he was tapped. The fluid had become encapsulated and shut off in one portion of the abdomen; this fluid was then withdrawn and it has never returned. The patient has remained well ever since, and please note, also, that he worked for nearly a year with that fluid in his belly. That is clinical fact number two. Clinical fact number three is that certain cases seem to recover after surgical interference, and probably by means of it Which cases are these? This is an important practical question. A case comes under observation: how is it to be treated? Is it a case for surgery? Certainly my knowledge is not sufficient to enable me to state to you clearly those cases which are amenable to surgery, and to surgery alone. I do not know, and I do not believe that any one does at present. It is a question for future solution. Perhaps this is as good a statement as I can make,—surgery should be resorted to in those cases of tuberculous peritonitis in which no improvement takes place after a fair trial of other means, and in which the tuberculosis is not so widespread as to render it practically useless to interfere at all.

There has been a good deal of speculation and discussion as to how surgery is curative. It was suggested that it was either by the draining off of the fluid, or by the admission of air to the peritoneal cavity, or by both, that the injurious influence on the bacilli was exerted. As far as drainage goes, this might explain the cases where recovery takes place after tapping, but we see cases recover without interference of any kind. The admission of air to the peritoneal cavity: it cannot be that, because that supposition does not explain the cases that get well spontaneously or the cases that get well after tapping alone. Of course, the peritoneal cavity may get into such a state from chronic inflammation that it loses its absorptive power. I suppose that was the case with our patient: the cavity over here on the right was probably tied up so that no absorption could take place. Whether it would have done harm to leave the fluid there we do not know; but certainly it did no harm to take it out, and the patient approaches more nearly the standard of a normal man without it than with it.

So that the chief result of surgery in this line is to demonstrate the curability of peritoneal tuberculosis. Modern surgery has also shown us that many of the cases of peritoneal inflammation formerly classed as primary or idiopathic are really tuberculous. There seems to be a tendency among some pathologists to be sceptical as to the tuberculous nature of

cases ending in recovery. It is asserted that the nodules in these cases are fibrous. We must recognize the fallibility of the naked eye. I recall a case in my own wards about two years ago. A young man was under treatment for abdominal pain and tenderness, accompanied by fever and distention of the peritoneal cavity with fluid. I believed the symptoms to be due to tuberculous peritonitis, and, after careful consideration, advised laparotomy. When Dr. Beach opened the abdomen the diagnosis seemed to be confirmed, as the peritoneum was everywhere studded with miliary nodules, with some larger masses which looked like the cheesy masses of agglomerated and older materials. Microscopic examination, however, showed the disease to be cancer, and the patient died soon after. To my mind, at least, the evidence is very strong that tuberculous peritonitis gets well, and I think our patients should receive the benefit of this fact in prognosis.

To return to the patient before us. He has been well now for four years, but his tuberculosis may some time start up again. There may be some bacilli bottled up in him which may get into the blood or lymphatic current, and he may go off rapidly; but he has been perfectly well, and it is not necessary that anything else should come.

The second important lesson suggested by this case concerns the natural history and prognosis of tuberculosis in general. Koch's discovery of the bacillus of tuberculosis has given us a criterion for tuberculosis. It has enlarged our conception very much; it has not only enlarged, but it has defined, our conception of tuberculosis; it has shown us how wide-spread tuberculosis is; it has brought together lesions which were formerly separated more or less; it has brought lupus and scrofula into the same category as consumption. People saw that there was some relation between scrofula and tuberculosis, but what that relation was they were not able to say. We recognized the fact that people with scrofulous glands were especially prone to consumption. The great Hebra, who was a physician before he was a dermatologist, used to say that perhaps scrofula and lupus were syphilis in the third generation. Koch's discovery has confirmed and extended Laennec's views as to the unity of tuberculosis.

Now, almost any organ or tissue may be the seat of tuberculosis, and tuberculosis may be general or it may be local. Let us take first the local forms. It seems to me that the gravity of the local form depends practically upon two things: it depends upon the tissue involved, or it depends upon the seat of the lesion. I mean in any given case the gravity depends upon where the tubercle is. There are certain portions of the body and certain tissues which afford a much better breeding-place for the bacilli than others; in certain parts it is more easy for diffusion to take place through the blood- or lymph-current, so that the disease may become generalized. The second thing which determines the gravity is the character of the tissues of the individual, the vulnerability of the individual, or the soil which he furnishes for tuberculous growth. We recognize the very

great difference which exists in different individuals in this respect. We see some people who will acquire pulmonary tuberculosis with the greatest ease, because, as we explain it, they furnish a favorable soil for the growth of the bacillus; we see others who become tuberculous only with the greatest difficulty, or perhaps are unable to become tuberculous. We see the same in animals: some, as the guinea-pig and rabbit, easily become tuberculous; others, as the dog, show great resisting power to the bacillus. We know that about 1.7 of all deaths, at all ages, in what we call civilized countries, are due to one form of tuberculosis,—namely, tuberculosis of the lungs. Now, the lungs form an ideal breeding-place for the bacilli; they are delightfully warm and moist, and full of nice little chambers and nooks, within which the bacilli can nestle, and if one house gets crowded there are plenty more in the neighborhood ready for occupancy. If you were going to contrive a tissue favorable for the breeding of the bacillus of tuberculosis, you could not produce a better one than the lungs; and the lungs, moreover, are easy of access. Patients are spitting around everywhere, the sputum is dried, and we are all breathing in its contained germs continually. The wonder is, not that so many people die of consumption of the lungs, but that so few die; it is a wonder that there are so many of us left, considering the opportunities for infection. We know that tuberculosis of the lungs gets well; we have just as good proof of this as we have of the fact that we are here in this room this morning. I should like to know how many people have had tuberculosis of the lungs and have never known it: I believe plenty of us have had consumption and have never been aware of it,—a diminutive crop or colony, perhaps, which was choked out and disappeared. I can see no escape from that view when we consider how many patients have recovered whom we know have had the disease. We see cases with abundant physical signs of the lesion and with bacilli in the sputum; we see them live on, and we see them well. Then from the pathological side we see on the autopsy-table scarred and puckered pulmonary apices with cheesy nodules, perhaps, in subjects who have had no history of cough or hæmoptysis: we thus have pathological evidence of cure. We have to recognize the fact that many cases get well, and the question naturally arises, How many persons have had pulmonary tuberculosis and have never known it?

We have touched upon pulmonary and peritoneal tuberculosis; now let us for a moment consider meningeal tuberculosis. *A priori* one might say that the membranes of the brain do not afford so favorable a breeding-place for the bacilli as do the lungs, for instance, however it may be with the peritoneum. The danger here lies in the absolute importance of the brain to life and the predilection of the disease for the base of the brain where the centres of respiration and circulation are found. It is stated in the books to-day, and it is the general belief of the profession, that tuberculous meningitis is necessarily fatal; that if you make a diagnosis of tuberculous meningitis and recovery takes place you may be sure that your diagnosis

was wrong. I am not convinced of the truth of this view. Demonstrative proof in such a case is most difficult; but experience as well as anology make me sceptical, or incredulous, if you prefer it so.

I recall the case of the child of a mother who was consumptive for some time before its birth and who is consumptive to-day, although, so good is her general condition, it would never occur to anybody that she had such a serious disease without a knowledge of her history and a physical examination. Shortly before and after the birth of the child her disease was more active than it has been since. She nursed the baby herself. During its second year this child had every symptom of tuberculous meningitis except convulsions. I held out no hopes of recovery; but the child got well and remains well to-day at the age of six or seven. The meningeal is probably the most dangerous form of tuberculosis, by reason of the narrowness of the cavity within which acute pathological changes may take place consistently with the preservation of life. But I firmly believe that recovery may, and sometimes does, take place. I think it was Koch himself who remarked that a bacillus is not a lion.

In genito-urinary tuberculosis we have the opportunity of demonstrating the existence and, perhaps later, the disappearance of the bacillus. But the time during which this branch of the subject has received adequate study is too brief to allow us to arrive at definite conclusions. We certainly see the disease here pursue a very chronic course.

To return for a moment to the lungs. A consumptive may either get well, or he may die of the disease; or he may live for many years, tuberculous all the time, and finally die of something else. A patient of mine, now nearly seventy years old, has had a cough for upward of twenty-five years, during at least twenty of which he has been under my observation from time to time. He is well nourished and it would never occur to you that he is a sick man if you should see him at rest. A trifling exertion, however, puts him out of breath. I have always considered his case as one of pure fibroid phthisis with emphysema and secondary heart changes. Recently I was led to examine the sputum as a matter of curiosity, and found abundant bacilli in it. As I look back at the case, its history, symptoms, and progress, I am convinced that his pulmonary disease has been tuberculosis from the start. I see no reason why he should not live ten years more, so well does he himself and those about him understand his limitations and management.

We have thus far considered local forms of tuberculosis. Let us turn for a moment to the general miliary form. Is it possible that recovery from this should take place? I well remember the absolute incredulity with which I read a paper by Dr. McCall Anderson, of Glasgow, some ten or fifteen years ago, in which he maintained the curability of general miliary tuberculosis and in which he adduced cases in support of his view. I wisely shook my head and mentally exclaimed "nonsense," or something to that effect. But to-day I am less incredulous. I am, indeed, prepared

to believe that he is right. Proof is difficult or impossible. But I can imagine that an individual may have acquired a local tuberculosis, limited in area, of longer or shorter duration, and that some of the bacilli may have got into the blood- or lymph-current and thus been more or less widely distributed over the system. Also that his power of resistance may have been such as to enable him to dispose of the organisms after a struggle, more or less prolonged and more or less intense. The amount of the disease and the vitality of the subject must count for much. Under certain conditions any tuberculosis may be recovered from.

If the facts warrant this view, and personally I think they do, our hands are strengthened in dealing with our patients. If you can feel that there is hope, however little, so much the better for your patient. It seems to me that the case which I have shown you to-day gives us just ground for encouragement of which you and your patients may fairly have the benefit. Twenty years ago we should have classed his glandular and rib disease as scrofulous and called his pleurisy and peritonitis idiopathic. To-day we do not talk much about idiopathic inflammation of serous membranes, or indeed of any other tissues.

REVIEW OF MEDICINE.

MEDICINE.

IN CHARGE OF JOHN H. MUSSER, M.D.,

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AND

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A Case of Exophthalmic Goitre, with Monocular Symptoms and Unilateral Thyroid Hypertrophy. (*Medical Record*, July 13, 1895.) By Percy Fridenberg, M.D., of New York.

The patient was a female, aged twenty-four years, who, after much worry and excitement from domestic causes, developed an increase of nervous excitability with the addition of palpitation, dyspnœa, and throbbing of the vessels of the neck. On examination decided exophthalmus of the left eye was noticed, together with marked Graefe's symptom and increase in size of the right lobe and the isthmus of the thyroid. In addition to these symptoms the patient presented a fine, fibrillary tremor in the tongue and hands, tumultuous heart's action, carotid pulsation, venous hum, radial pulse of one hundred and twenty per minute, superficial respiration, flushing and

hyperidrosis, and insufficiency of the recti interni muscles of the eye. A search of the literature reveals thirteen cases of Graves's disease presenting unilateral symptoms. The right eye was affected in all cases except three. The general improvement in the condition of the patient under consideration would indicate that the crossed condition will eventually recede with no change in the limitation of the symptoms.

History of an Epidemic of Exanthematic Typhus; Trial of Sero-Therapy; Beneficial Action of Subcutaneous Injections of the Serum of Convalescents from Typhus. (*Gazette des Hôpitaux*, July 4, 1895.) By Dr. E. Legrain.

The epidemic started in the civil prison of Bougie, in the province of Constantine, in November, 1894; on the 20th of November there were ten fully-developed cases. The prison was built in 1803 on the site of a cemetery; and from 1809 to 1816 all the parts to the west of the city were used as burying places for a large number of Kabyles who had died of a pest. There is a question whether typhus could renew its activity after such a long time. The epidemic assumed very serious proportions, the Europeans being the greatest sufferers. Serum was prepared from two convalescents from grave typhus, and three patients were injected with two, four, and six cubic centimetres respectively. The temperature fell in all in proportion to the quantity of serum injected; the injection of two cubic centimetres giving a drop of 1.2° C. A case is reported that had been for two days in a desperate condition: profound stupor, temperature-curve high and without remission; marked pericardial effusion; pulse thready and irregular, 135 per minute; toxic hemiplegia; respirations 52 per minute. No relief was produced by cold baths or by painting with guaiacol. An injection of ten cubic centimetres of serum was given, and in five hours the temperature had fallen 2° C.; pulse 100, full, and nearly regular; the pericardial effusion had diminished in considerable degree; the urine became abundant, and the respiration became more calm, almost normal. This benefit was not lasting, and in forty hours a second injection of twelve cubic centimetres produced a permanent amelioration of the symptoms. The fall of temperature was not sudden; in taking the temperature every two hours it was noticed that the drop commenced three hours after the injection, and reached its maximum at the fifteenth hour. The cardiac phenomena are probably toxic and not organic affections of the myocardium or of the valves. In a certain number of cases the pulse was improved in a very short time after the injection; from thready it became full and more regular. Generally the slowing of the pulse kept pace with the fall of the temperature. In several cases the coma disappeared in eight or ten hours after the injection of the serum. A case of left-sided hemiplegia, evidently of toxic origin, disappeared in eight hours after the injection. As a rule, the injections have very sensibly relieved the state of patients suffering from grave typhus, when the injection is made in the

first days of the disease. The use of the injections should be reserved for these severe cases.

Actinomycosis cured by Internal Medication with Potassium Iodide, and without Surgical Interference. (*Mercredi Médical*, June 19, 1895.) By A. Poncet.

An interesting history of a case of actinomycosis of the cheek and temporo-maxillary region is related by Poncet. The patient refusing all surgical interference, a complete cure was effected by internal medication with potassium iodide in doses of four grammes per day, continued for over six months. The method of contracting the disease seems to point strongly to the habit which the man had, in his walks in the country, of holding small pieces of hay, straw, etc., in his mouth.

Tests of the Hæmoglobin of Pregnant Women. (*New York Medical Journal*, June 22, 1895.) By Ethel Blackwell, M.D., of New York.

In endeavoring to answer the question, Are pregnant women anæmic? the author estimated the hæmoglobin present in one hundred and sixty-three pregnant women. The examinations were made uniform as to the locality from which the blood was taken and as to the time of making the examination. The majority of these women had an average of between forty and fifty per cent. of hæmoglobin, the general average being 54.2. These results were compared with those obtained from the examination of the blood of forty women students at Bryn Mawr College, one-half of whom had between eighty and ninety per cent. of hæmoglobin; this was distinctly an anæmic condition. When, however, it was remembered that the pregnant women examined belonged to the servant class of New York, the question arose, was this an anæmic condition when compared with non-pregnant women of the same class? An estimation of the hæmoglobin in one hundred non-pregnant women of the same class was made, and a general average of 53.1 per cent. was found. Twenty-three women were tested in the eighth and ninth months of pregnancy and again within twelve days after delivery. The general average of the præpartum cases was 60.5 per cent. and of the postpartum cases 53.2 per cent. Twenty-eight tests were made under six days after delivery, with an average of 53.2 per cent. Twenty-seven cases were examined from six to twelve days after delivery, with an average of 56.8 per cent. At the time of estimating twenty-one postpartum cases the babies' hæmoglobin was also estimated. In sixteen of these the babies' blood showed an average of 96.3 per cent., and the mothers' showed 49.6 per cent., while six of these babies had over one hundred per cent. of hæmoglobin. In a case of twins, the babies had 80.7 per cent. and 81 per cent. respectively, while the mother had 44.7 per cent. In another case the larger child was born first and the cord was immediately tied; he had sixty per cent., the smaller child had eighty-four per cent., while the mother had thirty-nine per cent. at the same time. In

the case of a baby born suddenly of a blind mother, the cord was torn, allowing hemorrhage; the hæmoglobin was forty-five per cent. in the baby and 34.5 per cent. in the mother. Some estimations were made to determine the daily variations of hæmoglobin in the blood of pregnant women. The results gave variations of from three-tenths to seventeen per cent. The results of this investigation may be thus summarized:

1. Compared with non-pregnant women of the same class, the hæmoglobin of the pregnant woman is higher in proportion; 53.1 per cent. in the former, 54.2 per cent. in the latter.

2. The percentage of hæmoglobin falls immediately after delivery.

3. There is a higher general average in the later days after delivery. The exhaustion and loss of blood during the first few days after labor will probably explain this.

4. There is a higher per cent. of hæmoglobin in the blood of the child than in that of the mother.

On the Clinical Value of Ehrlich's "Diazo"-Reaction. (*Medical Times and Hospital Gazette*, July 20, 1895.) By William J. M. Ettles, M.B.

After an examination of two hundred and forty-three specimens of urine, the author concludes that it is found in the urine of a certain number of apparently healthy individuals, two out of eighty-five cases. It is found invariably in typhoid fever and pneumonia. It is found very frequently in pleurisy, twelve out of thirteen cases; in measles, eight out of sixteen cases; and in intestinal obstruction, one out of two cases. It is found frequently in peritonitis, two out of five cases; in suppurative inflammation, four out of sixteen cases; in erysipelas, three out of seven cases; and in phthisis, five out of twenty-one cases. It is occasionally found in rachitis, three out of fourteen cases, and in diabetes mellitus, two out of seventeen cases. It seems to be absent in malignant and chronic, non-tuberculous, visceral lesions. When taken in connection with other symptoms it has a diagnostic importance. Its presence would turn the balance of opinion in favor of a case being measles instead of rötheln. Where we are suspicious of typhoid fever its presence would still further incline us to that view, and it would help differentiate tuberculous phthisis from other chronic, pulmonary diseases. These results agree with Nissen's deductions.

Goitre in Michigan. (*Medical News*, July 20, 1895.) By George Dock, M.D., of Ann Arbor, Michigan.

Goitre occurs in all parts of Michigan; it is most frequent in the northern part of both peninsulas, and in the southern part of the lower peninsula. It develops most frequently at about puberty; it affects females in larger proportion than males. In most cases the goitres are small; there is comparative symmetry in the growths, and the grotesque shapes, so

common in Switzerland, are rarely seen. No histological examinations have been made; but the tumors have all the physical characteristics of various forms of beginning struma. Exophthalmic goitre is unusually prevalent in Michigan; but with no evident relation to simple goitre. Goitre affects the lower animals almost always where it is common in man. Horses seem most frequently affected, then dogs, calves, and lambs. In accordance with current knowledge, the pathogenic substance causing goitre is supposed to be a micro-organism or a toxin; but nothing definite is known in this connection. In the cases occurring in Michigan, a very large proportion so afflicted have used well water. No case of simple goitre has been seen in which there was a murmur, even in rapidly-growing tumors. In case of Graves's disease, from which exophthalmus is absent, this sign can be used with advantage. The cases treated with dried thyroid are too few for the drawing of conclusions, but in young persons the remedy seems useful and should be further tried. In addition to improvement in the surroundings of the patient, the sterilizing of the drinking water and the removal of the patient to a non-goitrous district should be tried. The case of a man, aged sixty-six, is reported. This patient had a goitre which had grown in six weeks, and which had involved both lobes and the isthmus of the thyroid gland. After gramme doses of thyroid powder three times a day, continued for two days, the remedy was of necessity suspended, and in a week the goitre had disappeared together with its troublesome symptoms, leaving the neck measuring only one-half inch more than normal in circumference.

A Study of Obstinate Hiccough. (*Dublin Journal of Medical Science*, January, 1895.) By W. Langford Symes, L.R.C.S.I., L.R.C.P.I.

An old gentleman, aged eighty-seven, of a gouty disposition, became afflicted with obstinate hiccough during convalescence from an attack of congestion of the lungs. All remedies were tried with no avail, and the attack finally stopped suddenly after having persisted ten days and nine nights. During this time it is estimated that two hundred and fifty-seven thousand five hundred and twenty spasms occurred. Hiccough is a reflex spasm of the diaphragm, with subsequent closure of the glottis. It may be due to inflammatory diseases of the viscera; to irritation of these viscera; to specific causes, as malaria and gout; or it may be purely neurotic in origin. The pathology is believed to be a reflex spasm of the diaphragm with simultaneous closure of the glottis; having as the afferent nerve the pneumogastric and as efferent nerves the phrenic and recurrent laryngeal. It does not seem clear, however, that the phrenic nerve does transmit the efferent impulse. From clinical observations the sympathetic connections of the semilunar ganglion seem more likely to convey the impressions, for the following reasons: The diaphragm appears to contract before the laryngeal muscles, pointing to a closer and more direct communication with the gastric portion of the vagus than even the recurrent laryngeal.

The course of the phrenic nerve is healthy, and its respiratory function is perfect.

The patient has no control over the spasm, while the phrenic nerve is always subservient to one's will.

Remedies applied to the origin or course of the phrenic or to the cervical spine, such as blisters, ice-bags, compression, etc., have no effect; while those directed to the diaphragm, stomach, and solar plexus are generally curative.

The connection between the pneumogastric and phrenic nerves by means of the third, fourth, and fifth cervical nerves is remote, and if this were the route taken the impression must travel more than twice as fast on the phrenic as it does on the recurrent laryngeal nerve, since it reaches the diaphragm before it does the larynx,—conditions which are unphysiological.

The experiences of Romberg and of Bright show that direct irritation of the phrenic will not produce hiccough.

The existence of a perfect reflex loop between the stomach and diaphragm answers the purpose, is separate from the function of respiration, and is beyond the control of the patient.

The spasm is influenced by the acts of deglutition or vomiting to a greater degree than by any respiratory efforts.

The prognosis is questionable. Much depends on the cause of the spasm and much on the age and the condition of the patient. The treatment may be divided into the empirical, the antispasmodic, and the physiological.

THERAPEUTICS.

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D. J. EVANS, M.D.

The Use of Chlorine in the Treatment of Typhoid Fever. (*American Journal of the Medical Sciences*, September, 1895.) By Reynold W. Wilcox, M.D., LL.D., of New York City.

Wilcox has found the aqua chlori of the *United States Pharmacopœia* an efficient means of administering chlorine, but its solution must be freshly prepared. Chlorine-water should contain at least four-tenths of one per cent. of the gas, and, well diluted, can be administered in doses of from one to four fluidrachms every two, three, or four hours. As soon as the fæces lose their offensive odor, and the odor of chlorine is perceived in them, the dose-limit has been reached, and this amount continued will be sufficient.

The writer has reserved chlorine for cases which have come under observation after ulceration has been established and systemic infection has become marked. In cases seen early, where the manifestations are pre-

sumably local, the insoluble intestinal antiseptics, as naphthalin, salol, bismuth salicylate, beta-naphthol bismuth, and tribromophenol bismuth, are indicated. In the four cases cited, which are considered as being average ones, and illustrate very well the effect of the administration of chlorine, chlorine was employed after other methods, as cold bathing, calomel, hydrochloric acid, had failed. In the treatment of typhoid fever,—

1. Chlorine can be safely administered until complete disinfection of the alimentary canal is obtained.

2. Under its use the tongue becomes cleaner, the appetite and digestion better, fever lower, and the stools devoid of odor.

3. The general strength, the intellectual processes, and nervous conditions improve.

4. The disease is shortened in duration, and usually proceeds to rapid and complete recovery.

On some New Medicines in the Therapeutics of Tuberculosis of the Lungs. (*Therapeutische Wochenschrift*, August 25, 1895.) By Professor E. de Renzi, M.D., of Naples.

The author reports on the effect of some new medicines used in the treatment of tuberculosis as well as of some older medicines the doses of which have been modified.

He first speaks of acidum phenosuccinicum, phenosuccin, and iodophenosuccin. The trials on animals and patients showed that these three preparations do not influence considerably the tuberculous process. There was also no constant action on the fever.

Nuclein was given in parenchymatous injections, beginning with one-half cubic centimetre and one-fourth cubic centimetre daily, up to large doses of three and one-fourth cubic centimetres. These injections caused neither local nor general troubles. In most cases they were well borne. Of five patients treated, three showed marked general improvement, one showed considerable decrease of râles, but no great improvement.

Creosotal, creosotum carbonatum, was given three patients in doses of one-half to thirty grammes per day for nine, twelve, and thirty-four days. In all cases it was well received. There was an improvement in all, one even with increase in weight. The fever was not influenced by this medicine; the average temperature was even some tenths higher during treatment.

Hæmoglobin nardi was given to six tuberculous patients in doses of one to ten grains per day for three to seventeen days. Also with this medicine no remarkable effect was obtained, the general condition improved, however, in some, especially when they had taken large doses.

Aqua oxygenata had no effect: the average temperature remained the same.

Liquor arsenicalis was tried in large doses on four patients, increasing from four to fifty drops per day for nine to seventeen days. No trouble was observed from the large doses; the weight increased in all cases. There

was, however, no further general improvement. In one case there was noted decrease in the râles.

Electrical light of an arc lamp of fifty volts and of an intensity of thirty to fifty milliampères was thrown by a forty-centimetre reflector direct on the thorax of the four patients. In order to neutralize the effect of the heat an electrical ventilator was used. The patients in general stood the ordeal well. In all there were noticed decrease of temperature and of the number of bacilli, although the general condition and the lungs did not show much change. The exposition lasted from thirteen to fifty minutes. It results from these trials, which were somewhat difficult, that if one should succeed in using a very intense light, without calorific rays, considerable curative effects might be obtained.

The thermophor consists in a very long silver wire, which is coiled up between two woollen pieces so that the whole can be applied to the thorax. When the current is turned on the temperature increases considerably. This thermophor was used on six patients, who stood it well, even with high temperature. This was between 60° and 90° C. under the influence of the thermophor on the thorax. Three of the patients improved visibly. One who was treated thirty-four days increased three kilos in weight, and the râles decreased.

The Antisudorific Properties of Picrotoxin. (*Lancet*, September 14, 1895.) By M. Semmola, M.D., of Naples.

The author believes with Goltz, Kendall, and others that there are special sudoriparous nerves independent of the vasomotor system, while the presence of sudorific centres has been demonstrated experimentally in the cat by Vulpian, Luchsinger, Nawrocki, Ostruomon, Marmé, and Adamkewiez. Before an antisudorific treatment be instituted it is necessary to form a clear conception of the pathogeny of the disturbance in the secretion, and, according to whether the nervous sudoral system, the vasomotor system, or the smooth fibres of the blood-vessels, etc., enter into play, one must choose that pharmaceutical substance which, when absorbed, can best modify and counteract the disturbances which, in that especial patient, have caused the diaphoresis. When treating an ephidrosis due to excitement of the sudoriparous nervous system, we must have recourse to the medicines, like agaricine, atropine, etc., which have an inhibitory effect, thus compensating the increase of function; on the contrary, when we believe the ephidrosis is caused by paralysis of the vasomotor centre, we should administer exciting remedies, such as picrotoxin; and, lastly, if there be a diminution of vascular tone from peripheral disturbances, we then use hydropathy, which has such a marked action on the parietes of the blood-vessels.

To a patient with insufficiency of the mitral valves and great prostration following influenza and ephidrosis and cutaneous patches, picrotoxin was administered. Scarcely three days after the remedy had been given

(in doses of half a milligramme twice a day) the sweats gradually diminished, and entirely disappeared on the eighth day of treatment, while hitherto they had been obstinate against all other remedies.

Semmola considers that he was dealing in this case with a perspiration due to paresis, more especially caused by some of the many toxins which are often superadded to influenza, and which exerted their action on the sudoriparous centre and the vasomotor centre of the bulb. The livid red marks on the skin were certainly due to vasomotor paresis, and so confirmed the diagnosis.

SURGERY.

IN CHARGE OF HENRY R. WHARTON, M.D.,

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AND

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Successful Removal of a Large Tumor from the Spinal Canal. (*Beiträge zum Centralblatt für Chirurgie*, No. 27, 1895.) By Dr. Kümmell, of Hamburg.

The author relates an interesting case in which a tumor was diagnosed and removed from the spinal canal of a patient, aged forty-seven, with almost entire recovery and relief from pain. The patient had complained, in 1889, of weakness and pain in the limbs which kept gradually increasing, and which resisted all treatment. In May, 1893, a tumor was discovered on the inner surface of the rectum and successfully removed, the patient recovering entirely. This tumor was found to be a sarcoma. The following year the patient again had gradually increasing pain and weakness in the lower extremities, with localized pain beneath the shoulder-blades, followed by a paralysis. On the 6th of November of that year the patient was unable to move at all. The area of anæsthesia extended to the third thoracic vertebra on the left side and to the seventh on the right, with an area of hyperæsthesia extending above. The situation of these areas and the history of their development, together with the history of previous malignant growth, made the diagnosis of a tumor in the spinal canal at about the third thoracic vertebra on the left side nearly certain, although there were no external evidences of its presence. The patient's condition made operation necessary, and an incision over the third, fourth, and fifth vertebræ disclosed an external roughening as an evidence of the presence of disease. The removal of the spinous processes and arches disclosed a crumbling, bleeding tumor within the canal, but not a part of the spinal cord, although the latter was compressed to a marked extent, in sharp

contrast to the normal size above and below the tumor. The compressed portion was dark red and markedly cyanotic. The tumor had no connection with the dura, from which it was easily separated, although a portion of the vertebræ had undergone pressure necrosis. The operation of removal of the tumor and cleansing of the wound occupied a little over half an hour. It is of interest to note the change that took place in the compressed portion of the cord during the short time it was under observation; it rapidly regained its normal size, lost the bluish rose-color, and differed only from the normal part of the cord above and below by the inflammatory color remaining in it. The patient sustained the operation well; the pain in the wound was very slight, and the day following the operation reflex convulsions manifested themselves in the lower extremities. There was, however, no marked decrease in the paralytic symptoms for the next fourteen days, although there was a gradual increase in the severity of the convulsions. The great toes of the right and then the left foot were moved on the sixteenth day after the operation, and the other toes on the succeeding days. A week then elapsed, when the patient moved first the right and then the left foot; other groups of muscles gradually regained their power, until, a month and a half after operation, the patient could raise his legs up from the bed. Sensibility improved gradually with the return of motion, and two months after operation the patient could stand, and later acquired the use of his limbs, so that he could walk for a length of time and climb up and down stairs with the aid of a cane. The only remaining effects of the paralysis are incontinence of urine and difficulty in defecation. The tumor was found to be a sarcoma about the size of an apple. The prognosis for the future of the patient is, therefore, unfavorable so far as return of the disease in some other situation is concerned; but the relief of the symptoms and paralysis make the operation justifiable and of great value to the patient.

Penetrating Wounds of the Abdomen. (*Boston Medical and Surgical Journal*, July 25, 1895.) By James G. Mumford.

A study of thirty-eight cases treated in the Massachusetts General Hospital during the past twenty-four years forms the basis of this paper. Nineteen of these were stab-wound and the same number were gunshot-wound cases. All of the first class were operated upon, of which three unusually serious cases died,—the first of hemorrhage from opening of the external iliac artery, the second from laceration of the spleen, and the third from wounding of the omentum and stomach, causing hemorrhage and extravasation of stomach contents. The remaining sixteen cases recovered. In fifteen there was no penetration of the alimentary track; in one perforation of the intestine took place, but, although there was extravasation of fæces, the general peritoneal cavity remained unpolluted.

Of the gunshot victims, twelve of the nineteen died. Seven were operated on, and in every case the viscera were perforated, the deaths being

due to extravasation from the stomach and intestines, to injury of the kidneys, or to a wounded aorta. As with the stab-wounds, in every case in which there was extravasation into the general peritoneal cavity the patient died. Of the seven cases that recovered, five had no perforation and two perforation of the stomach only. The relatively lower death-rate after wounds of the stomach, as compared with those of the small intestine, may be accounted for by the anatomy of the parts. Both the stomach and the large intestine contain more muscle than the small intestine, and the various coats are not so closely associated. The mucous membrane is much more loosely attached and acts like a valve when a small hole is punctured, preventing extravasation. The colon is in part extraperitoneal, and the contents being less liquid than in the small intestine do not easily escape when the bowel is punctured.

The study of a very large number of cases convinces the author that nearly all extravasations are fatal. Stomach wounds with extravasation are fatal; without, they are generally recovered from. The same is true of the small intestine; but extravasation follows most small intestine wounds, while it does not follow most stomach wounds. A low death-rate from hemorrhage is due to early checking by operations. From wounds of the kidney the mortality has been high of recent years. Most of the fatal cases were brought in in a state of collapse and died in a few hours. The collapse in the majority of cases was not caused by hemorrhage but by extravasation.

To sum up this paper in brief,—

Gunshot-wounds show a much higher mortality than stab-wounds, but those stab-wounds which penetrate the viscera show a mortality equally high.

Wounds of the small intestine are usually fatal. Wounds of the stomach may be survived.

Explore all doubtful cases. This is the course of least danger.

Barring hemorrhage, early death after the accident is not from shock but sepsis; because,

Finally, this is seen only when the fatal extravasation takes place.

Intestinal Invagination and its Treatment. (*Beiträge zum Centralblatt für Chirurgie*, No. 27, 1895.) By Dr. Rydygier, of Krakau.

This author has gathered together a series of eighty-four new cases of operation for this condition from reliable sources; and from his study of them and cases occurring in his own practice draws the following conclusions:

In acute intestinal invagination,—

1. Operation should be employed as soon as non-operative measures have been thoroughly tried without success.

2. After the laparotomy disinvagination should be attempted before all other methods. If there are any suspicious-looking points on the intestinal

wall where rupture may be feared, the peritoneum should be walled off with iodoform gauze, or the part should be drawn out of the abdominal cavity.

3. When disinvagination is impossible, resection of the invaginatum should be employed, through an incision in the wall of the invaginator.

4. The resection of the entire invagination is to be employed where there are marked alterations in the wall of the intestine and perforation threatens.

5. The formation of an artificial anus and entero-anastomosis have usually no place in acute intestinal invagination. Only in case of collapse is an *anus præternaturalis* made admissable.

In chronic intestinal invagination,—

1. Non-operative therapeutic measures should be employed, but not for more than one week.

2. During the period between the attacks, operation is to be advised.

3. After laparotomy disinvagination is to be attempted tentatively. Four successful cases have been reported,—one at the end of nine months, two after six months, and one after ten weeks.

If this does not succeed, resection of the invaginatum should be employed. Entero-anastomosis may be employed where there are adhesions; but there are no indications in chronic intestinal invagination for the formation of an artificial anus.

NEUROLOGY.

IN CHARGE OF LANDON CARTER GRAY, M.D.,
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GUY HINSDALE, M.D., Philadelphia, AND WILLIAM BROADDUS PRITCHARD, M.D., New York City.

Causes Conspiring to Produce Mental Enfeeblement in Children; Causes of Imbecility; Varieties of Idiocy; Pathology of Idiocy. (*American Medico-Surgical Bulletin*, August 1, 1895.) By F. Savary Pearce, M.D., of Philadelphia.

1. Causes of mental backwardness. These causes may be divided into those operating before and those operating after birth. Among the causes operating before birth may be mentioned inebriety, syphilis, and phthisis, shock and nervousness. The percentages are necessarily inaccurate because parents and relatives keep back information as to the first three and give prominence to the last two. Phthisis is a very frequent predisposing cause. Among the postnatal complications may be mentioned accident, abuse and neglect, disease, sunstroke, and instrumental delivery. Of these, fever should receive more attention, since prolonged high temperature must have a deleterious influence on the developing brain. 2. Varieties of imbecility

or idiocy. As one classification we find congenital idiots, children born with impaired mentality; accidental idiots, children in whom trauma, epilepsy, etc., are causes; and developmental idiots, children in whom the mental enfeeblement comes on in an apparently brilliant child at some special epoch of strain in its life history. According to the somewhat vague resemblance of idiots to the different races we have the ethnic types of idiots of Langdon Down: Mongolian, Caucasian, Negroid, Ethiopian, Malay, Kalmuc, and American Indian. There are also the mercurial idiot and the "idiots savans." Ireland's classification is based on the causes conspiring to idiocy or existing with it, on the anatomy, or on certain movements assumed by the idiots. We have, then, the choreic, cretinoid, eclamptic, epileptic, genetous, hydrocephalic, hypertrophic, microcephalic, inflammatory, paralytic, plagiocephalic, scaphocephalic, strumous, taxic, the idiot of deprivation, and the sensorial idiot. 3. Pathology of idiocy. In considering this subject we must remember the rapidity of the development of the brain at different ages, and we must consider whether the pathological cause acts early or late. Anæmia is rare as an uncomplicated pathological entity. Hyperæmia, likewise, is not common as an unity; but, when it does exist, the dura and pia are loaded with blood, as are the sinuses and the choroid plexus. Atrophy of the brain may be local or general, and when partial is congenital or acquired. In this condition the nerve-cells are round instead of stellate and are not branched. In the white matter few nerve-fibres exist as compared with excess of neuroglia or connective-tissue elements. There may be shrinking of the anterior cornua of the cord; but no degeneration in its white columns has been noted. Hypertrophy is rare and is general or partial and is to be told from hydrocephalus. Virchow describes it as an excess of the neuroglia or connective tissue, which breaks down and becomes the granular-looking *débris* described by Rokitansky. Softening often occurs, either locally or generally, and is due to less blood being carried to the brain. Meningitis, tumors, and thrombi of the middle cerebral arteries or of the superior longitudinal sinus may be potent causes of softening. Microscopically, remnants of nerve-fibres, granular cells, *débris*, and blood-corpuscles are seen throughout the field. Sclerosis may be diffused in spots no larger than a pin's head or in patches. Here, again, the medullary substance is increased as compared with the gray matter. Hydrocephalus is seen in not a few idiots, and it is in these cases that the extremities are frequently paralyzed from excessive cerebral pressure. Porencephalus is either the result of diffuse hemorrhage *in utero* or is due to trauma later in life. Tumors may occur and are either tuberculous or gliomatous. Asymmetry of the head and of the convolutions is sometimes found. Alteration of the proportion of white to gray matter in the brain, when existent, consists of a great increase of the white substance. Simplicity of the convolutions in size and arrangement is often seen and is an evidence of reversion to our arboreal ancestry. Chronic meningitis is a frequent pathological condition. Acute meningitis is often

the immediate precursor or the cause of death, and is either simple or tuberculous. The most frequent lesions of the cerebellum are atrophy, tuberculous tumors, and cysts. Degenerative changes are found in the cord where cerebral palsies coexist with imbecility. As deformities we most frequently find high palatal arches, badly set teeth, and club-foot. The latter is more common in males. Histologically, the nerve-cells are round, not pyramidal, the nucleus is round or oval, and is in the centre with retracted protoplasm around it, thus leaving a clear space or a faintly-stained granular substance. Only occasionally are the nuclei at the base of a cell; at times the nucleus is at the apex of the nerve-cell. The nerve-cells have few processes and these are small and stunted. The apical process is nearly always present. Now and then processes exist but no spaces can be found. Where sections are hardened there is often noted a curious honey-comb condition, due to the number of cells with spaces seen in the field. In frozen sections no clear spaces are to be seen. The nucleus stains deeply, the protoplasm less so. The apical process of the nerve-cell is always seen and the cells are less stunted than in hardened sections. In either method of examining the cortex not more than five layers are detected, and the third layer is most affected.

The Knee-Jerk in Locomotor Ataxia, complicated by Hemiplegia.—M. Raichline reported at the Society of Biology, Paris, June 29, 1895, a case observed with M. Landrieux, in which secondary contracture and restoration of the tendon reflexes occurred after an organic hemiplegia, in a person afflicted with tabes. This is an unusual event, as usually a hemiplegia remains placid and the reflexes do not return if the person afflicted is already tabetic. He considered the loss of the knee-jerk in tabes as not due to a destruction of the reflex arc, but that the disease exerts an inhibitory influence on the reflex activity of the cord, and that due to the secondary degeneration of the crossed pyramidal tract, this reflex activity is exaggerated, and the knee-jerk is restored in some cases.

A Case of Tabes, with Involvement of One Spinal Root, in General Paralysis. (*Revue neurologique*, numbers 12, 13, and 14, June and July, 1895.) By Dr. Nageotte, chief of the Laboratory of Professor Raymond at the Salpêtrière, Paris.

In the sacral portion of a cord in a case of general paralysis Dr. Nageotte has found the interstitial neuritis of the roots, which we have described in a previous number of the INTERNATIONAL MEDICAL MAGAZINE. The nerve substance proper was not involved at this level of the cord, as the lesion had not existed sufficiently long. There was a uniform meningitis everywhere. A slight degeneration was noticed in the pyramidal tracts, which increased from above downward, and began at the medulla oblongata.

At the level of the lower fibres of the second and the upper fibres of the third dorsal roots a slight sclerosis was found against each posterior horn in the column of Burdach, touching the gelatinous substance of Rolando, and passing towards the point of entrance of the roots. In the same region there was a diminution of the fibres in the zone of Lissauer and in the posterior horns. These bands of sclerosis in higher sections passed inward towards the internal part of the columns of Burdach in the form of crescents. Below the third dorsal there was a degeneration of the descending branches of the nerves affected, which diminished in extent, and became separated from the posterior horns at lower levels. There are only three cases of lesion of isolated roots of the upper part of the cord recorded (Pfeifer, Gombault, Sottas) and in none is a descending degeneration described.

Dr. Nageotte has found the lesion discovered by him in the cords of six patients who had died from general paralysis, and he believes that the pure form of tabes and the form seen in general paralysis are the same. He asserts that the theory which places the primary lesion in the spinal ganglions lacks anatomical foundation, and that the theory of toxins which act on the posterior cord is incapable of proof.

Obersteiner and Redlich speak of a meningitis and an arterial sclerosis as the cause of tabes. In the case described by Dr. Nageotte only a few intramedullary fibres were diseased, and at this point the meningitis was not greater than elsewhere; the spinal roots showed no change. Obersteiner and Redlich have claimed that the posterior roots are not affected in beginning tabes, and have explained this by putting the seat of primary lesion at the point of entrance of the roots into the cord. When the roots are later affected it is due to retrograde degeneration. No one has described a retrograde intramedullary degeneration of the posterior root fibres; are we to consider that the extramedullary portion is more sensitive? Retrograde degeneration of the pyramidal tract is known, but one cannot compare the different columns of the cord too closely. Obersteiner admits that the meningitis is not always found in tabes. In many cases of spinal syphilis the meningitis is of a high grade, and yet there is no tabes. Careful study of the arrangement of the pia fibres at the point of entrance of the posterior roots shows that strangulation is impossible. Nor does any vessel enter the cord at this point. In a longitudinal cut the slightest obliquity can increase the appearance of the ring-like contraction of the pia at the point of entrance. The cord is elastic, after removal from the vertebral column, and after three days of hardening it shortens one-tenth of its previous length. The roots are also contractile. All this produces the appearance of stricture around the entering roots, but this was not present in a cord hardened by Dr. Nageotte without removal from the vertebral column. The author claims that the root neuritis, described by him, compresses and interferes with the nutrition of the root; that this latter commences to degenerate in the cord at the part farthest from the trophic

centres, while the root itself may appear normal. In general paralysis the degeneration is also greatest in that portion of the pyramidal tract which is farthest from the cerebral cortex.

OPHTHALMOLOGY.

IN CHARGE OF CHARLES A. OLIVER, A.M., M.D.,
Philadelphia, Pennsylvania,
AND
EDMUND LANDOLT, M.D.,
Paris, France.

A Case of Paralysis of the Superior Recti Muscles. (*Columbus Medical Journal*, May 28, 1895.)

A most interesting and instructive instance of this exceedingly rare type of ocular disturbance has been seen by William E. Bruner, of Cleveland, Ohio. The peculiarities of the case consist in the facts that there was intermittency in the relative strengths of the two paretic muscles; that there was nothing to indicate spasm of the inferior oblique, and that there were two degrees of esophoria.

The author, inclining to the thought that the paralysis in his case was congenital in origin, tends to the belief that the condition was due to a central deficiency rather than to malformation or arrest of development of the muscles.

Diphtheritic Conjunctivitis; its Treatment with Antitoxin Serum. (*Annales d'Oculistique* (English edition), April, 1895.)

Without being able to assert positively that spontaneous recovery could not have occurred in four most interesting and typical cases of diphtheritic conjunctivitis which were successfully treated by injections of antitoxin serum, V. Morax, of Paris, believes that the amelioration of the local changes and the betterment of the general condition so quickly followed the use of the material that its therapeutic usefulness must be assumed.

In doubtful cases where there is a pseudo-membrane with purulent discharge, he supplements the serum injection by the employment of a collyrium of nitrate of silver. Where the case, though certain by bacteriological study to be diphtheritic in nature, does not present any such secretion, the serum injection alone, he thinks, is sufficient to produce resolution and recovery.

Gallicin, a Gallic Acid Derivative; its Use in the Treatment of Eye-Diseases. (*Annals of Ophthalmology and Otology*, July, 1895.)

The results obtained by the use of this preparation of gallic acid, which was first employed in some two hundred ophthalmic cases by Mellinger, of

Basel, have been confirmed by George F. Suker, of Toledo, Ohio, in an additional series of thirty cases. He sums up the therapeutic value of the drug as follows:

It is applicable in all cases of catarrhal affections of the mucous membrane of the eye, either with or without secondary eczema, in cases of phlyctenular keratitis or conjunctivitis, and is exceedingly serviceable in follicular conditions and in superficial keratitis.

He believes that it is indeed worthy of trial, and in the future will, no doubt, play an important *rôle* in the treatment of the above-named diseases. He thinks that it is best employed in the powdered form; and, being very light, he has found that about one centigramme is sufficient for an application. He says it is to be applied once or twice daily as may be deemed expedient.

Alveolar Fibro-Sarcoma of the Optic Nerve. (*Archives of Ophthalmology*, April, 1895.)

Finlay, of Havana, Cuba, has had the good fortune to study the histological appearances of such a growth which occurred in the practice of Lopez, of the same city. As he says, it was curious to note the difference in structure presented by the tumor at different points. He found that it was instructive to follow the transition from the more highly-organized alveolar sarcomatous condition to the more atypical ordinary variety, and into the still more lowly-organized myxosarcoma.

From a careful analysis of the statistics of the results of operative procedure in such cases, he believes that operative interference is called for as soon as the diagnosis is made, as the tumor, if left to itself, will almost necessarily produce death, and the earlier the operation is performed the greater the chance of success. He thinks that "the comparatively large proportion of deaths immediately after the operation makes a strict adherence to the rules of aseptic and antiseptic surgery a necessity, and that the presence of metastatic growths within the cranium or of a direct intracranial extension seems to predispose to infection of the meninges; if such an extension can be inferred, all operative interference, with a radical cure in view, is contra-indicated."

He says that, in view of the good statistical returns, an attempt at preserving the eyeball is justifiable, and even recommendable, provided an ophthalmoscopic examination permits one to infer that there is no extension of the growth into the interior of the eye, and that the presence of the eyeball does not interfere with the complete excision of the tumor by limiting the field of operation.

He believes that where there is any suspicion of an extension into the orbit an exenteration should be performed, and says that the results of operations on recurring growths are not encouraging, but thinks that such a procedure may be at times beneficial, provided that there are no grounds to suspect an intracranial extension.

OBSTETRICS AND GYNÆCOLOGY.

IN CHARGE OF RICHARD C. NORRIS, M.D.,

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Some Practical Points in Connection with Rupture of the Female Perineum. (*Provincial Medical Journal*, July 1, 1895.) By William Dunnett Spanton, F.R.C.S.

The writer has operated on women of all ages between sixteen and seventy-three, and has found that age makes very little difference in the result of the operation. He thinks that elderly women should share the benefits which this safe procedure affords. With reference to the legal aspect in a case of laceration of the perineum left to itself, a case is cited where the physician neglected to repair the laceration immediately after its occurrence, in which septicæmia followed. An action was entered against the surgeon for damages, which resulted in the payment of the sum claimed by the patient, under the advice of the medical man's solicitor. He says, "The legal point is perfectly clear, that when in the ordinary course of treating a patient a mishap does occur, the surgeon will not be held liable provided he takes the necessary steps to put it right; but, on the other hand, the neglect to do this, whether from oversight or from a sense that it is unnecessary, renders him liable for any after-trouble, loss of time, and so on, to which the patient may be in consequence subjected."

The mode of operation adopted by the author is as follows: The patient is prepared by clearing the bowels several days before the operation, and an enema is administered a few hours previously, not just before the operation. The patient is kept in bed a day or two before the operation. The time preferred is just following a menstrual period. The surface is shaved and cleansed with carbolic soap and chloroform. The amount of denudation necessary will depend upon the extent of the tear. Usually the line between the labia minora and labia majora is a good guide, and a rounded incision is made across the septum formed by the remains of the perineum, and extending on each side far enough forward to form a broad surface, of which the sides are to be brought into apposition. The flap is dissected up by a so-called splitting process, keeping as nearly as may be equidistant from vagina and rectum. This flap of vaginal mucous membrane and skin is held forward and brought together and held there until the deep sutures have been inserted. A full-curved needle is passed from one side of the raw surface to the other, commencing about one-third of an inch outside the skin margin and emerging on the opposite side at a corresponding point. These silkworm sutures are placed about three-eighths of an inch apart, are drawn up, not too tight, and secured. Catgut sutures are then placed in the anterior vaginal margin and cut off short. If the rectum is involved,

a flap similar to the anterior one is made, commencing at the anterior anal margin. This flap is turned back, and a sharply-curved, thin Hagedorn needle carries a fine catgut suture first through one side and then the opposite, from the raw surface through into the rectum, leaving the ends hanging out. These are inserted about one-sixth inch apart, and are then tied from above downward, each knot being left in the rectum. An additional suture is placed at the point which is to represent the posterior edge of the new perineum, which helps to secure a rounded surface when all heals up.

PATHOLOGY.

IN CHARGE OF ALLEN J. SMITH, A.M., M.D.,
Professor of Pathology in the Medical Department of the University of Texas, Galveston, Texas,
AND
HENRY W. CATTELL, A.M., M.D.,
Demonstrator of Morbid Anatomy in the University of Pennsylvania; Prosector to the American Anthropometric Society; and Pathologist to the Presbyterian Hospital, the Institution for Feeble-Minded Children at Elwyn, etc.

On the Classification of the Amyloid Substance among the Albuminous Bodies. (*Zeitschrift für physiologische Chemie*, p. 343.) By A. Tschennak.

The amyloid material used in the experiments was in the form of a light yellow powder, and was obtained from amyloidically degenerated organs (liver and spleen) through complete mechanical pulverization and extraction by hot water, by alcohol, by ether, and by sieves. The material so prepared dissolved readily in alkalies, less so in organic and mineral acids, and on heating with water it is digested by pepsin and trypsin. There probably results at first unchanged but dissolved amyloid material, then it becomes an albuminate, and after longer action primary and secondary albumose and peptone. All these products gave the color reaction in the same way as the original substance, in part even more clearly. In regard to its elementary composition the amyloid material would therefore seem to belong to the albuminous bodies. In view of its comparatively lessened solubility, especially in acid, and its finer way of appearing in the organism, Tschennak counts the amyloid among the coagulated albuminous materials. The same place is given by the author to the hyaline substance, and he is of the opinion that the amyloid substance is a modified coagulation form of the circulating albumen, probably of the serum albumen, and not of the fibrin. The coagulation might take place in affected organs or in the already coagulated substance, and it might be conducted, perhaps through the leucocytes, from the blood to the organs and there deposited, eventually to be still further changed. Nothing could be established in regard to the cause of the remarkable color-reaction of the amyloid material.—*Centralblatt für medicinische Wissenschaften*, August 24, 1895.

A Quick Method of filtering Blood Serum. (*Johns Hopkins Hospital Bulletin.*) By Gwin Campbell, M.D., and A. D. Ghiselin, M.D.

The design of this filter was taken from an apparatus for filtering liquid through a Chamberlain bougie that appeared in the Army Exhibit at the World's Fair, in Chicago.

The filter proper is a single bougie water-filter strengthened to allow the safe use of high pressure, and arranged so that a flask may be attached without risk of contamination. A drum consisting of a cylinder four feet long by four inches in diameter which contains ten pounds of liquefied carbon dioxide is connected to the filter. In the upper part of the cylinder is a safety-valve and also a valve by which the pressure can be turned on. Attached to the latter is a thick-walled rubber hose which has fixed in it a pressure gauge registering three hundred pounds. A rubber-stoppered flask having two tubes passing through the stopper is used for collecting the filtrate. One tube is short, with an expanded end which is packed with cotton, and to the other long tube is attached a piece of rubber hose about two feet long, divided in the middle, and the two pieces joined by a glass nozzle. On this piece of hose are two of Mohr's pinchcocks. The other end of the hose is slipped over the open end of the bougie. After sterilizing this portion of the apparatus the bougie is introduced into the shell and the lower cap screwed on. The serum is poured in the upper opening of the shell. The drum is now connected to the filter, and when a pressure of about two hundred pounds is observed by the gauge the valve is closed. One thousand cubic centimetres of serum can be filtered in five minutes. It is perfectly clear, coagulates at exactly the same temperature as unfiltered serum, and no appreciable effect can be noted on any toxin or antitoxin that may be present.

FORENSIC MEDICINE.

IN CHARGE OF LORENZO D. BULETTE,
Of the Philadelphia Bar.

A CASE OF IMPERFECT REGISTRATION UNDER THE NEW YORK MEDICAL ACT.

The circumstances under which ignorance of the law excused the failure of a physician to register according to law find illustration in a recent case in New York.[1] The physician, instead of registering his license with the county clerk, filed it with the board of health; but, after the commencement of a prosecution to recover the penalty imposed for failure to make

[1] Mayor of New York *vs.* Blank, 34 N. Y. S. 92.

proper registration, and before trial, the physician made legal registration, and it was held that such subsequent registration is a defence to an action for the penalty.

The medical act of New York requires, as a condition precedent to the right to practice medicine, that a license be registered with the county clerk; and, for practising without such registration, the person so offending shall forfeit fifty dollars to the county. But the act also provides that if one "whose registration is not legal, because of error, misunderstanding, or unintentional omission, shall submit satisfactory proof that he had all the requirements prescribed by law at the time of his imperfect registration, he may, on unanimous recommendation of a State board of medical examiners, receive from the regents under seal a certificate of the facts, which may be registered by any county clerk, and shall make valid the previous imperfect registration." Having, from ignorance of the law, filed his license with the board of health, instead of with the county clerk, the defendant pursued the practice of medicine; and thereupon this action was brought to recover the prescribed penalty. At the trial the physician produced a certificate by the county clerk that, since the suit, he had made due registration, in conformity with the statute. Upon this state of fact, the prosecutor moved for judgment, but the court decided for the physician and dismissed the prosecutor's complaint.

On appeal, Mr. Justice Pryor, delivering the opinion of the court, said, "Despite the elaborate and interesting argument of plaintiff's counsel, we are of opinion that the action may not be maintained. 'In expounding penal statutes, it is an established rule that the construction must be strict as against the defendant, but liberal in his favor.' And this canon of interpretation was applied by a very great judge to precisely such a statute as that in question,—namely, one imposing a penalty for exercising a trade without due qualification.

"It is contended that, as the penalty was incurred by the defendant's unauthorized practice, his subsequent qualification is ineffectual to its remission. The registration with the board of health, argues the plaintiff's counsel, was not an imperfect registration, but simply no registration. The statute allows an 'illegal' registration to be repaired, where the cause of it was 'some error, misunderstanding, or omission.' The case is within the very terms of the statute. Nor is it less consistent with the policy of the statute, which is to admit a competent physician to practice when, in good faith, he has endeavored to comply with its requirements. If that be so, still the plaintiff insists that the statute is not retroactive, and is inoperative to validate an invalid registration. This is just the effect it contemplates and accomplishes. By its terms the subsequent registration 'shall make valid the previous imperfect registration.' What is thus made valid? Obviously, the previous imperfect registration. If this be not the true construction, then the words are meaningless, because without them the correct registration would take effect at once, and authorize future practice. If so,

urges the plaintiff, a man may register whenever he pleases. Why not, if, seeking to obey the law, he has evaded it by error or inadvertence? The construction we adopt would be repugnant to the policy of the statute if the unauthorized practice of an incompetent physician were condoned by the subsequent registration. But, to obtain such subsequent registration, he must 'submit satisfactory proof that he had all the requirements prescribed by law at the time of the imperfect registration.' Having such requirements, what harm ensues from suffering him to supplement the defective authentication of his competency? The subsequent registration demonstrates his qualification from the beginning. Nor was the remedial operation of the subsequent registration in legalizing prior practice arrested by the institution of this action. As the suit is by the public for the benefit of the public, a repeal of the statute *pendente lite* would cancel the penalty. Where the action is by an individual for his own benefit, such repeal, any time before final judgment, would bar recovery of the penalty. *A fortiori* is the penalty revoked by an act which the statute imposing it provides shall remove the provocation of the penalty. Without a penal clause there cannot be a penal consequence.

"It is conceded that the defendant is a competent physician, in large practice; that his failure to comply with the law in the first instance was not intentional, but inadvertent; that, apprised of his mistake, he promptly corrected it; that at the trial his qualification was certified in legal form. Why, then, should he be chastised by a penalty denounced against imposture only? To give the statute such an effect would be equally against the settled rules of construction, the obvious policy of the law, and the equities of the individual." And the judges all concurred in affirming the judgment in favor of the physician, and mulcted the prosecutor in costs.

BOOK REVIEWS.

SKIASCOPY AND ITS PRACTICAL APPLICATION TO THE STUDY OF REFRACTION. By Edward Jackson, A.M., M.D. Philadelphia: The Edwards & Docker Co., 1895.

This is a modest little book, written, as its author says, to bring about the more general adoption of skiascopy (the shadow-test) as an essential part of the examination for ametropia. The modesty of its appearance should not, however, cause it to be classed with those manuals which are simply compilations of the general knowledge of their subject. Remembering the paucity of the literature on the shadow-test, and how little real knowledge of it is the common property even of ophthalmologists, this work must be accorded a much higher place. It is easy, indeed, to see that its pages are the result of much original thought and painstaking investigation.

Its success in the direction of its author's purpose will depend, in the first place,

upon the usefulness of skiascopy itself; and, secondly, on the clearness with which the book teaches the art of its application. The author asks a strong claim for the method when he says, "It is by far the most accurate objective test. The limits of its accuracy depend on details of its execution, and the skill and patience of the observer; but it does not require any rare natural qualifications to carry it, for many eyes, to the extreme limits of accuracy for subjective tests." If this be admitted, the necessity to the student of ophthalmology of a mastery of the art of using the test is imperative. A purely objective method of measuring the refraction, which is as accurate as the usual subjective method, dealing, as the latter does, in quarter diopters, is a thing no ophthalmologist can afford to neglect.

The majority of those who have mastered the method will not regard the author's claim for it as too strongly worded. In fact, the usual objections one hears to the shadow-test relate to difficulties in its application. Though these are, haply, many, the accuracy of the result would outweigh them all, even were they more than they are.

Turning then to the technique of the application of the test, we find about three-fourths of the book devoted to this part of the subject. The descriptions of the various manual operations are written in a simple, terse style, while every point of difficulty in the study or use of the test has received careful consideration. The twenty-six illustrations are nearly all original, and are all good. Those showing the appearance of the light in the pupil in the forms of symmetrical aberration and irregular astigmatism will alone give shape to what many a student has, so far, literally, "seen through a glass, darkly."

The book is opportune. The use of the test has passed the experimental stage. The optical principles involved, and the best methods of its practical application, its advantages and its limitations, have been studied and defined. The method can now be presented entire, and recommended with confidence to the student. Every one acquainted with the part Dr. Jackson has taken in its development will recognize him as peculiarly fitted to so present the subject. It is safe to predict that the book will at once take its place as the standard work on the subject,—a subject destined to grow in recognition for many years to come.

R. J. P.

BOOKS RECEIVED.

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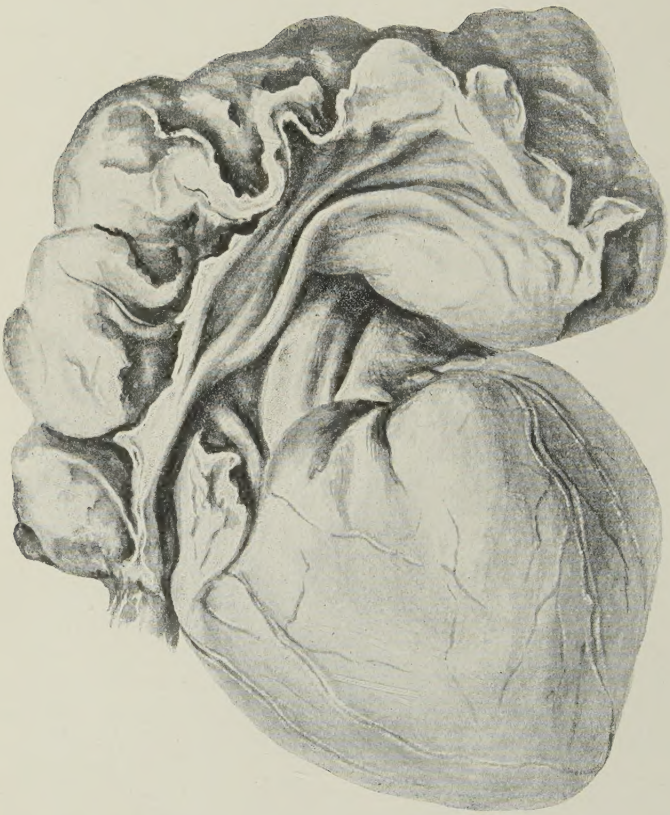
PLATE I.



Primary sarcoma of the thymus. The tumor covered the root of the great vessels and heart like a cap or bell. It is lifted upward in the illustration, showing the pericardium closely adherent to its under surface and exhibiting the roots of the great vessels and the heart below.